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IMPERIAL BUREAU OF ANIMAL NUTRITION

Technical Communication No. 15

MINERALS IN PASTURE DEFICIENCIES AND EXCESSES IN RELATION TO ANIMAL HEALTH

BY

F. C. RUSSELL, B.Sc.
Imperial Bureau of Animal Nutrition

WITH A FOREWORD BY

Sir JOHN BOYD ORR, D.S.O., M.C., M.D., F.R.S.

IMPERIAL BUREAU OF ANIMAL NUTRITION
ROWETT INSTITUTE, BUCKSBURN, ABERDEEN, SCOTLAND

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FOREWORD

PASTURE is the natural food of cattle, sheep and horses. It is the sole food of the majority of cattle and sheep in the world. Even under intensive methods of production where fodder crops and concentrates are fed, these are, on the whole, merely supplementary to pasture either grazed or in the form of hay, grass silage or dried grass. Pasture is thus the main raw material of milk, meat, mutton, wool, hides and other products of the herbivora. With the possible exception of cereals, it is the most important world crop, and, indeed, the misguided exploitation of large areas of land by cropping continuously with cereals has resulted in the major problem of soil erosion, the formation of wasting sores on the land surface that can best be healed by re-establishing pasture. It has been estimated that in the United Kingdom, in pre-war years, consumption of livestock products derived mainly from grass-lands amounted to about £400 million per annum compared with about £100 million for wheat. Grass-land products form a large proportion of the total exports of some countries—over 90 per cent. for New Zealand and over 50 per cent. for Australia and the Irish Free State. Pasture is, therefore, of great economic importance.

The importance of pasture will tend to increase in the future. As the standard of living rises, the consumption of dairy products, meat and mutton rises. In the wealthiest countries, the consumption of these per head is many times higher than in the poorer countries where the people subsist mainly on the cheap cereals. The World Food and Agricultural Conference of 1943 recommended that all governments should undertake the primary responsibility of bringing the diet of their people up to the standard needed for health. This will involve a great increase in the production of pasture products. In the United Kingdom, a committee of members of the House of Lords have estimated that the consumption of beef will need to be increased by 25 per cent. and of milk by 65 per cent. Taking the world as a whole, it will be many years before the products of pasture lands can be geared up to consumption requirements. There is thus a great need for an extension of research and experimental work on the improvement of pastures.

In the beginning of the present century, research on pasture problems was mainly by soil chemists and botanists. The former showed that pasture could be improved by the application of fertilisers, and the latter bred plants of higher feeding value and with different times of maturing so that a regular succession of plants at a stage of growth at which the feeding value is high is got throughout the grazing season. Great advances continued to be made along these lines. Early in the 1920's, however, a new line of investigation was opened up. The discovery of the vitamins led to a great expansion of work on the "deficiency diseases." Biochemists and physiologists began to investigate pasture from the point of view of the nutrition and health of the grazing animal. They took the clinical condition of the animal as the index of the nutritive value of the pasture. In the British Empire, generous grants for research were made by the Empire Marketing Board Research Committee. A great deal of pioneer work was done on the minerals known at that time to be essential for health by a team of workers drawn from the Rowett Institute at Aberdeen and the Animal Nutrition Institute at Cambridge and at Onderstepoort in South Africa. This new line was expanded and developed at Aberystwyth and many other centres in the British Empire and the United States. It has been found that mineral deficiencies in pasture are more important than vitamin deficiencies. If the pasture contains all the inorganic nutrients needed for health and is sufficient in quantity, there is unlikely to be a deficiency of either vitamins or protein. This early work emphasised the importance of the balance between soil and herbage and between the herbage and the grazing animal. A soil deficient in inorganic constituents and water cannot sustain good pasture no matter what the quality of the species of pasture plant sown and, in the same way, a pasture deficient in quality or quantity cannot support healthy animals. Loss has been sustained in attempts to grade up animals by improving the breed without first grading up the pasture to a nutritive level which will support the improved breed. The soil, the pasture and the grazing animal have all got to be considered together. Advances will be more rapid when pasture problems are tackled by a team of workers, including the soil chemist, the plant breeder, the nutritionist, the veterinarian and, not least important, those engaged in practical experimental work applying all scientific knowledge to the improvement of pasture and methods of conservation as hay, silage or dried grass.

This early work appeared to be of such importance that, in 1926, the Civil Research Committee of the Cabinet of the United Kingdom set up a sub-committee to consider and report on the relationship between the mineral content of pastures and their nutritive value. On the instructions of this committee, a review of the literature on minerals in pastures was prepared in 1928 and a supplementary review, dealing especially with the work being done within the British Empire at that time, was made in the following year.

Since the publication of these reviews, which dealt mainly with phosphorus, calcium, iodine and iron, there have been important developments in pasture research. One of the most interesting of these was in connection with trace elements. Work done in New Zealand had shown that crude iron salts helped to cure or prevent a type of disease found in various parts of the world. Later work in Australia and New Zealand showed that the cure was effected not by the iron but by traces of the rare elements present in the crude salts used. This opened up a new highly important line of research which has been prosecuted with vigour.

It is impossible to say whether the new work on cobalt and copper completes the story of deficiency of minerals in pasture. Further research may show that there are still more elements, the lack of which in pasture is the cause of malnutrition. Of equal interest are the results now being obtained on the presence of toxic mineral elements as, for example, selenium and molybdenum.

Much of this new knowledge obtained in the last ten years or so is now being applied with excellent results, but the volume of research is still meagre in view of the great economic importance of any increase of information which will lead to an increase in the nutritive value of pastures.

This is an aspect of the subject which is of special importance in the fight against disease. Recent advances in human medicine have shown that people suffering from even minor degrees of malnutrution are more susceptible to certain infectious diseases. This is true of grazing animals. Deficiencies in quality or quanity of pasture appear to predispose grazing animals to other diseases not directly attributable to the deficiencies. The veterinarian has, therefore, rightly come to take the leading part in work on mineral deficiencies and excesses in pasture.

It became obvious some time ago, that the great volume of literature which has appeared in recent years called for a new review of the literature on minerals in pastures. Miss Russell of the Imperial Bureau of Animal Nutrition was given the task and this publication is the result. Only those who have compiled and written such reviews appreciate the amount of laborious work needed to bring together all the available information from the many hundreds of papers published and arrange it in a logical and lucid form. Miss Russell has performed a service for which, I am sure, research workers in this field will be grateful.

12th May, 1944. J. B. ORR.

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CONTENTS

PAGE

FOREWORD iii

ACKNOWLEDGEMENTS iv

CHAPTER I.

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO DEFICIENCY OF COBALT

Introduction and Discussion 7
Bush-sickness. New Zealand 10
Morton Mains Disease. New Zealand 15
Mairoa Dopiness. New Zealand 18
Enzootic Marasmus. Australia 20
Nutritional Anaemia of Sheep. New South Wales 23
Nakuruitis. Kenya, Africa 23
Pining. United Kingdom 24
Grand Traverse Disease. Michigan, U.S.A. 28
"Neck-Ail." Massachusetts, U.S.A. 29
Cobalt Deficiency. Canada 29

CHAPTER II.

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO DUAL DEFICIENCY OF COBALT AND COPPER

Introduction 30
Coast Disease. Australia 30
Salt Sick. Florida, U.S.A. 33

CHAPTER III.

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO DEFICIENCY OF COPPER

Introduction and Discussion 35
Enzootic Ataxia. Australia 38
Swayback. United Kingdom 43
Swayback. Other Countries 46
Sudden Death or Falling Disease. Australia 46
So-Called Rickets or "Wobbles" in Foals and Calves. Australia and U.S.A. 49
"Stringy" or "Straight Steely" Wool in Sheep. Australia . . 50
Scouring Disease. Holland 50
Scouring Disease on "Teart" Pastures. United Kingdom . . . 51
Licking Disease Attributed to Copper Deficiency. Europe . . 54

CHAPTER IV.

DEFICIENCY OR IMBALANCE OF SODIUM, POTASSIUM AND CHLORINE

Licking Disease or Lecksucht 56

CHAPTER V.

DEFICIENCY OF IODINE
Goitre 63

CHAPTER VI

DEFICIENCY OF PHOSPHORUS OR CALCIUM 68

CHAPTER VII.

HYPOMAGNESAEMIC TETANY
Grass Tetany, Grass Staggers or Lactation Tetany 69

CHAPTER VIII.

SELENIUM EXCESS
Alkali Disease and Blind Staggers 75

CHAPTER IX.

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO MOLYBDENUM EXCESS
Introduction and Discussion 81
Scouring Disease on "Teart" Pastures. United Kingdom *see page* 51
Enzootic Haematuria of Cattle 82

CHAPTER X.

MANGANESE EXCESS
"Forage Anaemia" Associated with Infectious Anaemia . . . 82

CHAPTER XI.

FLUOROSIS. ARSENIC POISONING 86

CHAPTER XII.

NITRATE EXCESS
"Oat Hay Poisoning" 87

CHAPTER XIII.

GENERAL DISCUSSION 88
BIBLIOGRAPHY OF REVIEWS 90
APPENDIX. SUMMARY TABLE OF INVESTIGATIONS . . . *At end*

CHAPTER I

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO DEFICIENCY OF COBALT

INTRODUCTION AND DISCUSSION

The discovery that cobalt was essential for ruminant nutrition was made barely 10 years ago by investigators in Western and South Australia. For many years settlers in New Zealand and Australia had experienced severe losses of sheep and cattle from " wasting diseases " of unknown etiology. The trouble was confined to certain areas where sheep and cattle, after grazing for several months on " unsound " pasture, lost their appetite, wasted, and finally died of starvation, although surrounded by a luxuriant growth of pasture. Horses on the same pasture remained in excellent health. Spectacular recovery followed change to " sound " areas although the curative pastures often seemed very much inferior in quantity and quality to " affected " pasture. Unsound areas occurred on soils of diverse geological origin ranging from coarse volcanic soils in New Zealand to wind blown shell sands in South Australia. The periodical removal of cattle and sheep from " affected " to " sound " holdings proved to be a method of avoiding or cutting down stock losses but, since this was usually inconvenient and often practically impossible, determined efforts were made to discover the cause of the trouble.

The clinical picture of affected stock was indistinguishable from that of chronic starvation and gave no indication of the cause of the anorexia, but the anaemic appearance common in animals suffering from bush-sickness, as the condition was called in New Zealand, led Aston to try treatment with iron. He succeeded in curing bush-sick sheep by giving massive doses of iron compounds, and concluded that he was dealing with a deficiency of iron. Aston's successful treatment of bush-sickness in New Zealand led Filmer, who was investigating a similar disease, which he called enzootic marasmus, in Western Australia, to try the same treatment. Filmer, however, concluded from the massive doses of iron compounds that were needed to cure enzootic marasmus, and the variations in effectiveness of different crude iron compounds irrespective of their iron content, that the curative agent was not the iron but some impurity. This was confirmed by Filmer and Underwood in 1934. They found that a dilute hydrochloric acid extract of a sample of limonite which cured enzootic marasmus was also curative, and removal of the iron did not impair its activity. Further fractionation showed that the potent element was in the fraction containing nickel, manganese, cobalt and zinc. Tests with supposedly pure nickel oxide indicated that nickel was the curative element, but subsequent attempts to cure the disease with pure nickel chloride failed. Meanwhile Marston and his collaborators, investigating the wasting disease called " coast disease," which was enzootic in certain parts of Southern Australia, had formed the opinion that the condition was due to lack of one of the trace elements. One of these workers, Thomas, called attention to the polycythaemic action of cobalt for rats which had been demonstrated a few years previously by Waltner and Waltner and suggested that cobalt might be required in ruminant nutrition. At this stage discussion took place between Underwood of Western Australia and the South Australian workers, and subsequent investigations by Filmer and Underwood in Western Australia and Marston and his co-workers in South Australia established that the feeding of small quantities of pure cobalt salts was beneficial in both areas. The success previously obtained in treating enzootic marasmus with nickel oxide was found to be due to contamination of the oxide with cobalt. In the affected areas of Western Australia sheep were kept in excellent condition by a daily dose of 0·1 mg. cobalt, and cattle were cured and maintained in good health by administration of 0·3 to 1·0 mg. cobalt daily. In South Australia, however, it was found that copper as well as cobalt was necessary for complete recovery from and prevention of coast disease.

At the same time in New Zealand it had become clear from the work of Rigg, Askew, Grimmett and Shorland that the efficacy of certain ores and soil drenches in the treatment of bush-sickness was not related to their iron content. Askew and Dixon found that an extract of soil low in iron but containing cobalt, copper and nickel prevented the occurrence of bush-sickness. Following this line of investigation, which was also indicated by the Australian work, they established that cobalt alone was capable of curing and preventing the onset of bush-sickness and that the effectiveness of the ores and soil drenches was related to their cobalt content. Dixon also found that lack of cobalt was involved in a similar condition known as Morton Mains disease which affected lambs, especially before weaning, in certain parts of Southland. Sick lambs responded to cobalt feeding and the disease could be controlled by providing a cobalt lick for ewes and lambs. There is some evidence that the feeding of cobalt to

ewes during pregnancy has a beneficial effect on the health of lambs in these areas, and that cobalt can be transmitted through the placenta and stored by the foetus, but further investigations are needed to substantiate this.

Cobalt feeding has since proved beneficial in similar wasting diseases in other parts of the world. Cattle suffering from nakuruitis in Kenya, " pining " sheep in some parts of Britain, " salt sick " cattle in Florida and cattle suffering from a wasting disease in Michigan have responded well to this treatment. In Florida, however, as in the " coasty " areas of Australia deficiency of cobalt is associated with deficiency of copper.

From a purely practical viewpoint the problem of these wasting diseases was solved by the discovery that they could be controlled by the provision of cobalt in the form of a drench or as a cobaltised salt lick. But the implication that certain pastures might be inadequately supplied with cobalt to satisfy the needs of grazing sheep and cattle called for further investigations and, fortunately, in some affected areas, facilities for such research were available. The most systematic study of the problem was made in New Zealand, where it was clearly established that bush-sick animals were suffering from uncomplicated cobalt deficiency caused by lack of cobalt in the pasture. This conclusion rested on the evidence of cobalt feeding experiments, on the low cobalt reserves in organs such as the liver of affected animals compared with those of healthy or cobalt treated animals, and on the low cobalt content of affected pastures compared with healthy pastures. In general, pastures in affected areas had a low cobalt content, the critical values for sheep and cattle being respectively 0·07 and 0·04 p.p.m. D.M. of cobalt, and in areas where the pasture was low in cobalt at certain seasons only, the disease showed a seasonal incidence corresponding to the drop in the cobalt level of the pasture.

Analyses of organs of lambs in the Morton Mains disease areas showed low cobalt reserves in affected lambs, and analyses of samples of pasture on which lambs developed typical symptoms of the sickness showed a cobalt content of 0·04 to 0·07 p.p.m. D.M. Sufficient data are not available to show conclusively why cattle do not develop bush-sickness in this area, but from the investigations in the bush-sick areas it would appear that the explanation probably lies in the lower critical level of cobalt in pasture for cattle as compared with sheep. The critical level determined for cattle in bush-sick areas corresponds with the minimum recorded values in the Morton Mains area.

Fertilising with cobalt increased the cobalt content of bush-sick pasture, and annual application of less than 2 lb. cobalt chloride per acre proved an effective method of controlling bush-sickness, thus affording further confirmation that the disease was due to deficiency of cobalt in the pasture. The effectiveness of cobalt fertilising as a method of controlling Morton Mains disease has also been demonstrated.

Affected lambs in the Morton Mains district were shown to be heavily parasitised, but comparative trials with anthelmintic and cobalt treatment indicated that parasitic infestation, although it might be secondary to cobalt deficiency, was not primarily involved in the etiology of the disease.

In Western Australia also Underwood and Harvey demonstrated that the livers of sheep suffering from enzootic marasmus were comparatively low in cobalt, and correlated the incidence of the disease in sheep and cattle with the low cobalt content of the pastures which, in this area, falls as low as 0·02 p.p.m. D.M. Although the disease could be cured by cobalt administration, the livers of sheep cured of the disease were found to contain rather less than the normal amounts of copper. The copper content of " affected " pasture was found to be less than that of healthy pasture, and it was concluded that although copper deficiency was not concerned in the etiology of enzootic marasmus the copper content of these pastures was marginal for dry sheep and cattle and would probably be too low for breeding sheep. This will be discussed later under copper deficiency.

" Mairoa dopiness " in sheep in New Zealand, although in recent years looked upon as a cobalt deficiency disease and not distinguished from bush-sickness, has certain unusual features. Unlike bush-sick sheep, " dopy " sheep were found to have light bones with a low ash : organic matter ratio. Top-dressing the pasture with lime and superphosphate had a beneficial effect on the health of " dopy " sheep and improved the ash : organic matter ratio of their bones. The interpretation of these findings as due to deficiency of calcium has not been borne out by pasture analyses. McNaught claims to have obtained beneficial results with cobalt licks and has demonstrated that " affected " Mairoa pastures are as low in cobalt as bush-sick pastures. He also showed that fertilising with cobaltised superphosphate increased the cobalt content of Mairoa pasture and prevented the occurrence of the disease, but, although control animals on pasture treated with ordinary superphosphate did not show such good growth or wool yield, they remained fairly healthy over a period of a year. There is some evidence that fertilising with ordinary superphosphate increases the uptake of cobalt by pasture plants, and this might account for the lack of disease in the control animals, but Young's evidence that fertilising with ordinary superphosphate, which improved the ash : organic ratio of the bones of " dopy " sheep, raised the P_2O_5 content of the pasture from 0·41 to 0·84 per cent. seems to suggest that, in this area, cobalt deficiency may be complicated by a low phosphorus intake.

In the pining area of Easter Ross in Scotland the evidence of feeding trials, which indicated a deficiency of cobalt, has been confirmed by pasture analyses and the disease has been controlled by cobalt fertilising. No analysis of organs for cobalt has been published so far from this area. In other areas of Britain where "pining" has been attributed to deficiency of cobalt the conclusion rests mainly on the results of comparative trials of anthelmintic and cobalt treatment; such pastures as have been analysed for cobalt have not been sampled systematically to show seasonal variation, and no analysis of organs has been made for cobalt. It appears that the syndrome to which the term "pining" is applied may be caused by deficiency of cobalt in some areas, by parasitic infestation in other areas, and sometimes by a combination of the two. In Tiree the conditions under which pining occurs in cattle closely resemble those of "coasty" country in Australia and the disease may prove to be caused by a dual deficiency of cobalt and copper.

In Michigan the low cobalt content of hay from areas where cattle suffer from a wasting disease, known as Grand Traverse disease, tends to confirm the indications of cobalt feeding trials that the condition is due to deficiency of cobalt in the feed.

In Alberta, Bowstead *et al.* have shown that sheep confined to a diet consisting chiefly of locally grown non-leguminous hays of low cobalt content developed a wasting condition which proved fatal unless supplements of cobalt were given. Although there is no evidence that the condition occurs naturally in Canada, sheep in some parts of Alberta may be receiving insufficient cobalt for the maintenance of normal growth and thrift.

"Nutritional anaemia" of sheep in New South Wales and of cattle in Massachusetts have been included in this section because of the clinical descriptions and because of their response to iron ammonium citrate, but the role of cobalt in these conditions has apparently not yet been investigated.

It seems clear that the cobalt content of pasture is important for ruminant nutrition, but as yet the conditions under which cobalt deficient pastures occur are not fully understood. Cobalt deficiency disease appears on a variety of soils of widely different geological origin, such as the pumice and granite soils of New Zealand and the shell sands of South Australia, and recently McNaught and Paul (1940) have added a limestone soil in New Zealand to the list of soils which give rise to cobalt deficiency. There is some evidence that the cobalt content of soils is related to the magnesium content of the parent rocks, but the relationship between soil cobalt and pasture cobalt is not constant. In general, soils containing less than 2 p.p.m. of cobalt give pastures with insufficient cobalt for ruminant needs, but this is not always the case, and with any given cobalt level in soil, the cobalt content of pasture may be modified by the availability of the soil cobalt and the moisture relations of the soil, the stage of pasture growth, the species of pasture plant, and possibly manurial treatment.

Although it has been established beyond doubt that cobalt is essential for ruminants, the role which cobalt plays in ruminant nutrition is not yet known, nor is it understood why horses can do with less or possibly no cobalt in their diet. It was first suggested that cobalt deficiency might be concerned in the etiology of these wasting diseases because of the anaemic appearance of affected animals, and because large doses of cobalt had a polycythaemic action in rats. The diagnosis of anaemia was usually based on the pallor of the mucous membranes, but subsequent detailed investigations of the blood picture in bush-sickness gave no evidence of anaemia; in Morton Mains disease the degree of anaemia did not parallel the severity of the sickness; in coast disease anaemia as measured by haemoglobin and red cell count was not marked until the animals were *in extremis* and although, in general, "salt sick" cattle in Florida had a lower concentration of haemoglobin than healthy animals, a number of affected cattle showed high values. A less easily measured characteristic of these wasting diseases is a marked reduction of blood volume, and it is probable that changes in blood or plasma volume affect the concentration of haemoglobin to such an extent that haemoglobin determinations are not a reliable indication of changes in the ability of the animals to produce haemoglobin. For instance, feeding cobalt to sick animals often results in an immediate drop in haemoglobin level followed by a gradual rise to normal, and it is possible that this preliminary drop is due to the temporary inability of the haemopoietic system to keep pace with the sudden increase in plasma volume which follows restoration of appetite.

Absolute agreement has not, even now, been obtained regarding the results of feeding cobalt to experimental laboratory animals, but the concensus of evidence seems to be that a true polycythaemia can be produced in healthy animals given cobalt in addition to iron and copper. An attempt by Josland (1937) to produce polycythaemia in 6 healthy sheep by feeding very large amounts of cobalt was only partly successful. One sheep showed a marked polycythaemia as measured by increased concentration of erythrocytes and haemoglobin; one sheep was not affected and the remaining four became mildly anaemic. Josland suggested that the animals which became anaemic might have depleted their reserves of iron.

Frost *et al.* (1941) have pointed out that the polycythemic effect of cobalt may be quite unrelated to the essential role cobalt plays in the nutrition of certain species. They attempted to produce cobalt deficiency in dogs by confining them to a milk diet supplemented with iron and copper but

found that, although addition of small amounts of cobalt stimulated haematopoiesis in certain dogs in which the rate of blood formation appeared unusually slow, in many cases iron and copper sufficed with milk for normal haemoglobin formation. This was in agreement with the work of Underwood and Elvehjem (1938) on rats which showed that the increase in growth and haemoglobin regeneration in anaemic rats following addition of copper, manganese and cobalt-free iron to a milk diet was not improved upon by addition of 0·1 mg. cobalt daily. Analyses of the milk used in both these experiments indicated that the intake of cobalt from milk was of the order of 1·5 to 3 μg. per kg. of body weight per day in the dog experiments and about 6 μg. per kg. daily in the rat experiments compared with the estimated requirement of 4 μg. per kg. for a growing sheep. Frost *et al.* concluded that only the use of purified diets or of foods from cobalt deficient areas will permit a decision to be made as to whether cobalt is generally essential for the normal functioning of haematopoietic organs. Such haematological studies have been made on ruminants consuming foods low in cobalt, but since loss of appetite appears to be the first, or one of the first, effects of cobalt deficiency in ruminants, it is impossible to judge from the results of these studies whether cobalt deficiency has a specific effect on haemoglobin formation in the ruminant which is obscured by the results of failure to eat and drink, or whether anaemia when it occurs is merely secondary to inanition.

References to the investigations mentioned in this survey will be found in the more detailed accounts of the diseases attributed or attributable to cobalt deficiency which follow.

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BUSH-SICKNESS. NEW ZEALAND

Occurrence and Description of the Disease

This wasting disease occurs extensively among grazing sheep and cattle on the Central Plateau of the North Island and in the province of Nelson in the South Island. In the North Island the principal affected areas are located on the Taupo and Kaharoa volcanic showers which have given rise to coarse pumice soils, but all coarse volcanic soils are not necessarily bush-sick (Askew and Rigg, 1932). The disease has also been reported on the pumice soils of the Gisborne-East Cape back country and of Waikato, and on soils derived from older volcanic ash deposits in North Auckland (McNaught, 1938). The affected areas at Glenhope and the Sherry Valley, Nelson, are on granite soils, the former of coarse texture (Askew and Rigg, 1932). Unlike the coarse textured North Island and Glenhope bush-sick soils, affected soils in the Sherry Valley are loams (Askew, 1938).

The disease, as it occurs in sheep and cattle in the North Island of New Zealand, has been described by Reid (1923) as taking the form of a progressive anæmia accompanied by emaciation, loss of energy and death from exhaustion. The condition of the animals deteriorates despite an abundance of apparently good feed. The symptoms appear within about 5 months of pasturing on "affected" land, sheep being more susceptible than cattle, and younger animals more susceptible than older. Horses appear to be quite immune. *Post-mortem* examination shows a condition of extreme emaciation such as would occur in any case of chronic starvation. Askew and Rigg (1932) found the symptoms of bush-sickness in sheep at Glenhope on the South Island similar to those described by Reid. Frequently symptoms appeared within 6 weeks of transference to "affected" grazing. The skin and gums of affected sheep were pale in colour, and anæmia was stated to be a typical symptom of the Glenhope disease. However, in a detailed study of the blood in bush-sickness, Askew (1939 [1]) found no evidence of anæmia. This is discussed in more detail below. In some areas the disease may occur at all times of the year, while in others a seasonal variation is observed, the worst months being November and February. Hopkirk and Grimmett (1938) state that milk yield is adversely affected, probably because loss of appetite restricts feed intake.

Experimental Studies

Feeding experiments. When this subject was reviewed by Orr (1929), investigations into the etiology of bush-sickness in the North Island by Aston and his co-workers had shown that administration of certain iron salts, *e.g.* ferric ammonium citrate, both cured and prevented the onset of the disease. This was later confirmed for the disease as it occurred in Glenhope in the South Island (Askew and Rigg, 1932).

Further experiments by Aston (1932) focussed attention on limonite, a hydrated oxide of iron which was cheap and very effective in the treatment of bush-sick sheep and cattle. Drenching with Nelson soil was also found to be curative (Rigg and Askew, 1934). The work of Grimmett and Shorland (1934), Rigg and Askew (1934 and 1936), however, showed that all deposits of limonite were not equally curative, nor was the effectiveness of soil drenches and limonite related to their content of total iron or iron soluble in weak acid. Rigg and Askew (1936) found that an acid extract of Nelson soil which prevented bush-sickness was low in iron but contained cobalt, copper and nickel. Askew and Dixon (1936), continuing this line of investigation, which was also indicated as a result of contemporary work on wasting disease in Australia, were unable to cure bush-sickness in sheep with nickel or copper sulphate, but found that a drench of cobalt chloride providing 8 mg. cobalt per week was completely successful in overcoming bush-sickness at Glenhope. Analyses of limonites which had previously been used with success showed a cobalt content of 55 p.p.m. compared with unsatisfactory samples which contained only 2 to 12·5 p.p.m. Soils which gave curative drenches were also high in cobalt, containing from 17·5 to 43 p.p.m. Samples of ferric ammonium citrate and of an extract of Nelson soil which had proved beneficial but not curative each contained 24 p.p.m. of cobalt. It is suggested that the greater effectiveness of the soil containing 17·5 p.p.m. of cobalt may be connected with the availability of the cobalt.

Becker and Gaddum (1937) analysed spectrographically 2 samples of limonite from New Zealand, one effective and the other ineffective in correcting bush-sickness. They found in the effective sample 50 p.p.m. of cobalt but no trace of cobalt in the ineffective sample. They observed that the 2 samples had approximately the same copper content, and the ineffective sample had much higher contents of barium and strontium than had the effective sample.

The relation between the cobalt content of limonites and their curative value was later confirmed by McNaught (1937) for a large number of samples. He found a wide variation in cobalt content, samples from the Okaihau quarries alone ranging from 5·2 to 281 p.p.m. dry weight.

Following Askew and Dixon's (1936) cure of bush-sick sheep at Glenhope by administration of weekly doses of 8 mg. of cobalt as cobalt chloride, Wall (1937) found that daily dosing of bush-sick sheep in Rotorua with drenches of cobalt chloride supplying 7 mg. cobalt weekly was curative. Wall also observed that bush-sick sheep on the same pasture made excellent progress when given daily doses of limonite which provided only 1·7 mg. cobalt per week. An iron-free extract of the limonite which provided 0·4 mg. cobalt weekly was not effective. Hopkirk (1937) reported that the onset of bush-sickness on land on which control ewes and lambs developed the disease was prevented by administration of a weekly drench of cobalt sulphate providing 1·4 mg. cobalt for ewes and 0·7 mg. for lambs. At Glenhope, Askew (1939 [1]) found that weekly drenching of sheep with an acid extract or iron-free extract of soil prevented the onset of bush-sickness over a period of 2 years. The soil drench provided 0·4 mg. cobalt per week. Askew and Josland (1937), investigating the rate of excretion of cobalt in sheep drenched with 4 mg. cobalt, found that only about 2 per cent. of the cobalt appeared in the urine, mostly in the first 24 hours, the bulk of it being excreted in the fæces within 48 hours. They pointed out the importance of this result in drenching trials as an indication of the frequency with which this operation should be carried out if continuous absorption of cobalt is desired. The need for regular and frequent administration of cobalt has also been pointed out by Hopkirk (1937), who showed that although a weekly dose of 1·4 mg. cobalt for ewes and 0·7 mg. cobalt for lambs prevented the onset of bush-sickness, four times these doses given monthly were not very effective. This was confirmed by Filmer (1941), who showed that although the onset of bush-sickness could be prevented by monthly doses of 140 mg., cobalt, ewes and lambs receiving 1 mg. cobalt twice a week showed better liveweight increases.

Composition of the blood. In his early investigations into bush-sickness at Rotorua, Aston (1924) reported that the blood of bush-sick animals was extremely low in iron. Askew and Rigg (1932) analysed the blood of a Glenhope bush-sick sheep. They found low contents of iron, nitrogen and total solids compared with the average for 4 healthy Nelson sheep. Even allowing for the greatly lowered total solids, the percentage of iron was still considerably below normal.

Wall (1937), in her investigation of iron compounds and trace elements in the treatment of bush-sick sheep in the North Island, found that the values for total solids and total iron in the blood ran parallel and increased as the health of the animals improved. The copper contents of the blood of bush-sick sheep were within the range for normal healthy sheep in the district.

Askew and Dixon (1937 [1]) estimated the D.M., hæmoglobin and cobalt content of the blood of 6 sheep maintained on bush-sick pasture, 2 of which had been treated with 8 mg. cobalt per week, 2 with 8 mg. nickel per week from October, and 2 which had received no treatment. All the sheep had been on the property for 12 months, and sheep showing signs of sickness were distributed over the groups, 2 to the control and 3 to each of the nickel and cobalt groups. The results obtained in April were as follows :

	Control Sheep	Nickel Treated	Cobalt Treated
Hb: per cent. (Tallquist) . .	35–60	30–60	60–70
D.M. per cent. . . .	10·4–13·2	9·8–15·1	18·1–18·4
Co: p.p.m. D.M. . . .	0·02–0·02	0·02–0·01	0·05–0·04

It is pointed out that the cobalt content of the blood of the unhealthy animals was similar to the average for 9 samples from healthy sheep (0·02). The lower values for haemoglobin and D.M. in the control and nickel treated sheep were taken as indicative of a distinct tendency to anaemia in these poorly conditioned animals. Askew (1939 [1]) examined the blood of sheep which had been kept on unhealthy pasture for almost a year. The data gave no suggestion of anaemia, although pallor of gums and skin is a symptom of bush-sickness. No differences between values for haemoglobin (estimated by the Sahli method), red cell count, mean corpuscular volume and mean corpuscular haemoglobin were found between groups of sheep which had been kept in good condition by means of soil or cobalt drenches and a control group in which 1 animal had died and others were definitely bush-sick. The haemoglobin in g. per 100 c.c. for the former ranged from 8·6 to 13·0 compared with 10·4 to 12·1 f[illegible] the latter. This first examination was made in September, and by February, when the second exam[illegible] ination was made, none of the control animals remained in the experiment, they having died or been removed to healthy pasture because of bush-sickness, but the February results in the drenched groups did not differ appreciably from the September results. A third examination was made in November, and again showed little change in red cell count and haemoglobin. Between February and November, however, mean corpuscular volume had decreased and mean corpuscular haemoglobin had increased. The significance of these changes could not be assessed.

Composition of Organs. Askew and Dixon (1937 [1]) also analysed the organs of the unhealthy sheep and healthy cobalt-treated sheep on Glenhope bush-sick pasture, and found that the cobalt content of the liver (in April) was below normal in bush-sickness, 0·02 to 0·03 p.p.m. D.M. compared with a range of 0·07 to 0·25 for 9 healthy sheep on good pasture. The livers of the 2 cobalt treated animals had 0·23 and 0·22 p.p.m. D.M. The kidneys of sheep treated with cobalt had about six times and the gallbladder twice the amount of cobalt found in the kidneys and gallbladder of sick sheep. There was no appreciable difference between the cobalt contents of spleen and pancreas in the two groups. Askew (1939 [1]) analysed the livers of the sheep maintained in health for over 2 years on sick Glenhope pasture by means of soil or cobalt drenches, and found values of 0·05 and 0·09 p.p.m. D.M. in November. He concluded that a cobalt content of 0·05 p.p.m. D.M. was compatible with perfect health. The copper contents of the livers of these sheep were well within "normal" limits.

At Glenhope a trial was run in which sheep were kept on "affected" pasture until symptoms of bush-sickness appeared, after which the animals were treated with 8 mg. cobalt weekly. Periodically, animals were slaughtered and samples of liver, spleen, heart, kidneys and pancreas were obtained for analysis. As the animals became progressively bush-sick the cobalt content of all these organs decreased. After drenching began the cobalt contents increased greatly. Three weeks after th[illegible] treatment was begun, except for the spleen, the cobalt contents were almost as high as after fo[illegible] months of treatment. (Dept. Sci. Indust. Res. N.Z. 1943).*

Composition of the Soil and Pasture

Soil. McNaught (1937) analysed soils from the Rotorua district for cobalt soluble in decinormal hydrochloric acid, and found values ranging from 0·05 to 0·23 p.p.m. dry weight in bush-sick soils, and from 0·33 to 0·94 p.p.m. in healthy soils. Kidson (1937) estimated in a large number of soil samples from different areas in the North and South Islands the amount of cobalt soluble in concentrated hydrochloric acid. In general, it was found that soils where bush-sickness was prevalent had less than 2 p.p.m. of cobalt, while values for average healthy soils ranged from 6 to 20 p.p.m. Several soils, however, from areas where bush-sickness was not known were low in cobalt. The use of normal and decinormal hydrochloric acid, water and carbon dioxide instead of concentrated hydrochloric acid for extraction failed to dispose of these anomalies, and it was concluded that determination of cobalt in soils was not entirely satisfactory as a guide to the use of cobalt supplements for stock. Kidson (1938) pointed out that, in general, the cobalt content of soils is related to the magnesium content of their parent rocks. Results from a survey of the cobalt status of soils in the Sherry and Wangapeka districts of Nelson (Stanton and Kidson, 1939) indicated that the amount of cobalt soluble in concentrated hydrochloric acid in the soils depended to a large extent on geological formation, soils derived

* The detailed results have become available while this report was in press (Askew, H. O. and Watson, J. 1943, *N.Z. J. Sci. Technol.* [*A.*], **25,** 81).

from the Kaiteriteri granite being low in cobalt. Values of less than 2 p.p.m. of cobalt in the soil (dry weight) were generally associated with unhealthy stock.

Pasture. Aston (1924) analysed samples of red clover which he considered was probably the principal fodder of pumice lands, and showed that plants from the Rotorua bush-sick areas had a lower content of iron than plants from healthy areas. At Glenhope, Askew and Rigg (1932), investigating the three principal types of soil which occur, found that on each of the areas there was a distinct type of pasture. The Kaiteriteri (unhealthy) pasture consisted largely of fescue with agrostis spp. ; Moutere Hills (healthy) pasture consisted mainly of danthonia with agrostis, while a good mixture of English grasses and white clover was found in the Howard (healthy) area. Chemical analyses of samples of these pastures failed to explain the occurrence of bush-sickness on the Kaiteriteri soil and, contrary to Aston's findings, the distribution of bush-sickness was not reflected in the iron content of the pasture. As a result of the trend in soil investigations, attention was directed to the possibility of a deficiency of cobalt in bush-sick pastures. Investigations of the cobalt content of Nelson pastures carried out by Askew and his co-workers (Askew and Dixon, 1937 [2] ; Askew and Maunsell, 1937 ; Askew, 1938), and of North Island pastures (McNaught, 1938, summarised by Hopkirk and Grimmett, 1938) showed that, in general, bush-sick pastures were poor in cobalt compared with healthy pastures, the dividing line being about 0·07 p.p.m. D.M. in the pasture on both islands. McNaught observed that samples from several healthy areas were low in cobalt but, in general, his results indicated that North Island pastures containing less than 0·07 p.p.m. were unhealthy for sheep, while those containing less than 0·04 p.p.m. were unhealthy for sheep and cattle. Askew (1939 [1]) analysed bush-sick and healthy Nelson pasture for copper and found no evidence of a copper deficiency, which was in agreement with the results of liver copper studies and concluded that bush-sickness was a cobalt deficiency disease uncomplicated by lack of copper.

McNaughton (1938) found that the average cobalt value for 77 North Island pastures unhealthy for sheep was 0·038 p.p.m. D.M., and on the basis of a daily consumption of 2 lb. dry matter or 15 lb. fresh pasture he calculated that the weekly ingestion of cobalt by a sheep would be 0·24 mg. The average cobalt content of the 39 healthy pastures analysed was 0·106 p.p.m., giving a weekly intake of 0·67 mg. cobalt. The difference between these two (0·43 mg.) was approximately equal to that found by Askew and Dixon (1937 [2]) between healthy and unhealthy Glenhope pastures (0·35 mg.) on the basis of a daily intake of 1 kg. dry matter. Askew (1939 [1]) calculated from the analyses of Glenhope pasture upon which sheep became bush-sick, that the average weekly ingestion of cobalt during the season was 0·385 mg. per head. Sheep which received an additional supply of 0·4 mg. cobalt per head per week as a drench remained healthy.

Askew and Maunsell (1937) found a marked seasonal variation in the cobalt content of healthy Nelson pasture, their figures ranging in one pasture from 0·08 p.p.m. D.M. in May to 1·26 p.p.m. in September. McNaught, therefore, attempted to find an explanation of his apparently anomalous cases on the basis of seasonal variation (McNaught, 1938 ; McNaught and Paul, 1939). It was found that, in general, the cobalt content of pasture tended to increase in late autumn and winter when growth was retarded, and to decrease in spring and summer when growth was at its maximum. In a severely bush-sick area where sickness, especially in sheep, may be experienced at all times of the year, cobalt values, although reaching a minimum in December, were very low throughout the whole year. A more noticeable seasonal variation was observed in some other bush-sick areas, where the trend towards minimum values in summer corresponded with the seasonal incidence of the disease, the worst months being between November and February. In healthy areas the decrease in cobalt content with the flush of late spring growth was very rapid and, although low values might temporarily be found, it was considered that the reasonably high values obtaining over the greater part of the year carried sheep and cattle through the period of deficiency.

McNaught and Paul (1939), in this investigation of seasonal variation in the cobalt content of pasture, observed that in both cold weather and dry weather the cobalt content of pastures tends to increase. They also found evidence to suggest that close grazing increases the cobalt in the pasture, which may partly explain the beneficial results from rotational close grazing in cobalt deficiency areas.

Askew and Maunsell (1937), when they investigated the cobalt content of 3 healthy Nelson pastures on soils containing 41, 18 and 17 p.p.m. of cobalt, found that over a period of 9 months the cobalt content of pasture on the Nelson soil containing 41 p.p.m. of cobalt tended to be lower than at Appleby, where the soil contained 18 p.p.m. of cobalt. The former pasture consisted almost entirely of perennial rye grass, and the latter of perennial rye grass and white clover. The pasture on soil containing 17 p.p.m. showed the highest values for cobalt, and consisted mainly of white clover and perennial rye grass. In their survey of the cobalt content of Nelson soils and pasture sampled in March, April and May, Stanton and Kidson (1939) found that while, in general, the cobalt content of the pasture was correlated with that of the soil, particularly when the latter was low, this was not always the case. They suggested that the moisture relations of the soil, the availability of soil cobalt,

the stage of pasture growth, and the species of pasture plant, all play a part in determining the cobalt status of the pasture.

The relationship between the cobalt content of the soil and pasture and the incidence of bush-sickness is further complicated by the question of soil contamination of pasture. While the theory of iron starvation still held, Askew and Rigg (1932), in order to explain the lack of correlation between iron in the pasture and the incidence of bush-sickness, put forward the hypothesis that contamination of pasture with soil provides a large proportion of the iron required by grazing animals. Analysis of the paunch contents of a sheep grazing on unhealthy pasture, which presented few features favourable to contamination, showed that the soil ingested was not less than 2 per cent. on the dry matter basis of the pasture eaten. An early experiment of Gilruth (1900) was reinterpreted to support this new hypothesis. In it, sheep removed from a bush-sick area to a ploughed field free from vegetation in a healthy area remained healthy, although fed entirely on pasture cut from the unhealthy area, while a control group left to graze on the unhealthy pasture developed the disease. More recently, McNaught and Paul (1939) have pointed out that since cattle and sheep may benefit from the ingestion of soil drenches containing appreciable amounts of cobalt, contamination with soil may be important for the health of stock, especially in the winter months. Such contamination complicates the interpretation of analytical results, especially as it is not known to what extent soil cobalt is available to the animal, and makes it impossible to establish clearly defined standards for cobalt deficient and healthy pasture.

The cobalt content of the pasture can be increased by topdressing with cobalt salts. Askew and Maunsell (1937) showed that topdressing healthy Nelson pasture with 2 to 112 lb. cobalt chloride per acre gave marked increases in the cobalt content of the pasture. Annual applications of 2 lb. cobalt chloride per acre to unhealthy pasture to raise the cobalt content have proved an effective means of controlling bush-sickness (Askew and Dixon, 1937 [2]; Askew, 1939 [2]), and, indeed, experiments by Taylor *et al.* (1938), Askew (1939 [2]), and Bonner *et al.* (1939) indicate that a much smaller quantity of cobalt salt may suffice. Wright and Taylor (1931) found that application of 5 cwt. limestone and 2 cwt. superphosphate per acre to a reputed bush-sick area had beneficial effects on breeding ewes and their lambs as far as could be judged from an experimental period of 1 year. Askew (1934), however, observed that stock developed bush-sickness sooner on limed than on unlimed soil, and it was later shown that application of lime has a depressing effect on the uptake of cobalt by pasture (Askew and Dixon, 1937 [3]; Askew and Maunsell, 1937). This is dealt with in more detail on page 17.

In view of the findings of Mitchell *et al.* (1941) that fertilising pasture containing 0·07 p.p.m. of cobalt and 6·3 p.p.m. of molybdenum in a pining area of Scotland (see p. 27), with 2 lb. cobalt chloride per acre, raised the molybdenum content by about 50 per cent., it is pointed out in the *Dept. Sci. Indust. Res. N.Z. Ann. Rep.* (1943) that New Zealand pastures seldom contain more than 1 p.p.m. of molybdenum and fertilising with as much as 1 cwt. cobalt chloride per acre caused only a slight increase in the molybdenum content.

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MORTON MAINS DISEASE. NEW ZEALAND

Occurrence and Description of the Disease

A disease of grazing sheep, known as Morton Mains disease, has been reported by Dixon (1936) in several areas of Southland, including the Morton Mains district. A study of the soils in Southland revealed large areas of loessic soil, mostly of a silt loam texture, upon which both healthy and unhealthy farms were found.

The clinical symptoms, according to Dixon (1936), except for the lack of marked anæmia, were very similar to those of bush-sickness described by Askew and Rigg in 1932. It was considered, however, that these symptoms might merely be those of general malnutrition in sheep, and that the diseases might not be the same. Veterinarians, it was said, had shown that parasites were present in lambs in sufficient numbers to cause death. In Morton Mains lambs are the principal sufferers. The ailment usually appears about December in lambs which are still on their mothers, and the critical period appears to be from mid December to the end of January. After this time mortality is not so high and may even disappear, especially when the lambs have been weaned. Bell (1937) showed by means of blood studies on sick lambs that an anaemia, more pronounced as the season advances, often accompanies and accentuates Morton Mains disease in lambs but that the typical sickness can occur apart from any anaemia. She suggests that the anaemia is the result of prolonged malnutrition. This is discussed in more detail below. Histological study of the liver, pancreas, spleen and bone marrow showed no consistent difference between sick and healthy lambs.

Methods of management which in the past have proved beneficial are weaning, with subsequent change from grass to green feeds such as turnip tops or kale ; buying in fresh ewes each year and keeping grass short and fresh, *e.g.* by pasturing cows ahead of the flock. Dixon (1936) pointed out that all of these were consistent with either parasitic infestation or mineral deficiency as the cause of the sickness.

Experimental Studies

Feeding experiments. Dixon (1936) showed that drenching with limonite and certain Nelson and Bluff soils high in cobalt and copper had a beneficial effect on sick lambs. Further investigation (Askew and Dixon, 1936) showed that the element effective in preventing the sickness was one of the Gp. III B elements (zinc, manganese, nickel, cobalt) but since excellent results were also obtained with solutions of cobalt chloride it was concluded that the effectiveness of the soil drenches was due to their cobalt content. Twice-weekly drenches of soil providing 0·32 to 0·40 mg. cobalt weekly were satisfactory in preventing the ailment, a solution of Gp. III B elements prepared from the Bluff soil and providing 0·29 mg. cobalt weekly was not quite so effective, and 0·1 mg. cobalt weekly in the form of Otama soil extract did not give adequate protection. The best results were obtained with an aqueous solution of cobalt chloride providing 8 mg. cobalt weekly. Control lambs which were showing marked symptoms of sickness, and even lambs *in extremis* showed great improvement when given the cobalt chloride treatment. Dixon (1937 [1]) showed that the provision of a salt lick containing 2 oz. cobalt chloride to a ton of salt during pregnancy only, had a beneficial effect on the health of the offspring in affected areas, but better results were obtained when, in addition, a lick containing 4 oz. cobalt chloride per ton of salt was provided for both ewes and lambs after lambing. Later experiments by Dixon (1937 [2]) indicated that 0·16 mg. nickel incorporated into a weekly drench of 0·8 mg. cobalt gave better results than this amount of cobalt alone in the treatment of Morton Mains disease.

Dixon (1936) suggested parasitic infestation as a possible factor in the etiology of Morton Mains disease ; later experiments, however, showed that anthelmintic treatment with copper and nicotine sulphates was ineffective in preventing the sickness, but the disease could be prevented and cured by administration of cobalt chloride alone (Askew and Dixon, 1936).

Dixon (1937 [3]) pointed out that although Morton Mains and similar districts are generally regarded as free from cow or calf sickness akin to the lamb trouble, nevertheless, in view of Filmer and Underwood's experience in Australia that in areas of enzootic marasmus the growth of calves and the milk production of cows may be adversely affected without the appearance of more obvious symptoms, and that cobalt supplements had a beneficial effect on milk production in enzootic marasmus (p. 21), it seemed desirable to test the value of cobalt salts for dairy cows grazing on pasture at Morton

Mains. No beneficial effect on milk yield was observed when 4 mg. cobalt was given twice weekly to cows grazing in an area where lamb trouble had been experienced, but the experiments were not considered conclusive.

Composition of the blood. Bell (1937) examined the blood of lambs at Morton Mains where cobalt was effective in curing the ailment occurring in the district. The results are summarised in Table 1.

TABLE 1

Blood Picture in Morton Mains Disease

Date	No. of Lambs	Description of Lambs	Hb * g. per 100 c.c.	R.B.C. million per c.mm.
/12/36	2	2 months old—very sick, scarcely able to stand	13·8, 14·1	11·6, 9·8
	4	Not exhibiting marked symptoms of sickness	14·3	11·6
	6	Access to Co lick since birth, very good condition	13·9	11·1
	6	Not doing so well, but not showing any symptom	13·6	10·9
/2/36	2	Very sick	5·2, 6·9	6·3, 8·8
	9	Normal lambs at freezing works	12·0	—
	?	Receiving 8 mg. Co weekly	12·4	
/4/36	5	Receiving no supplement	10·3 (7·4 to 12·7)	8·8
	13	Receiving supplements of Co	13·4 (12·4 to 14·8)	10·7
	4	Exhibiting symptoms of sickness	7·9, 8·9, 9·3, 10·0	

* Hb values given as per cent., Sahli scale, have been converted to g. per 100 c.c. on the basis of 100 per cent. = 17·2 g. Hb.

Bell pointed out that the severity of the sickness does not run parallel to the degree of anaemia, and contends that the anaemia is secondary to poor nutrition and physical weakness, and is more likely to supervene as the season advances. Later, when the anaemia has developed, fatigue is more easily induced, with consequent reduction in grazing, and a vicious circle is set up.

In the feeding experiments reported by Dixon (Askew and Dixon, 1936) samples of blood from control and cobalt treated lambs were analysed for cobalt. The average for 3 untreated lambs was 0·01 p.p.m., and for 3 lambs receiving 8 mg. cobalt weekly was 0·03 p.p.m. D.M. (Askew and Dixon, 1937 [2]).

Composition of Organs. In these drench experiments (Askew and Dixon, 1937 [2]), Dixon found that the livers of control lambs, sick and potentially sick, were low in cobalt compared with those of healthy lambs from freezing works, and that cobalt treatment simultaneously restored bodily health and the cobalt reserve of the liver. The pancreas also appeared to store cobalt (Table 2).

TABLE 2

Cobalt in Organs of Sick and Healthy Lambs

Description of Lambs	No. of Lambs	Average Cobalt Content p.p.m. D.M.		
		Liver	Spleen	Pancreas
Healthy lambs from freezing works	2	0·17	0·06	0·04
Treated with 8 mg. Co weekly	4	0·20	0·04	0·07
Treated with soil extract containing 0·32 mg. Co weekly	2	0·04	0·015	0·02
Control, not doing well	4	0·025	0·03	0·025

There were also indications that cobalt fed to pregnant ewes could be transmitted to the offspring and stored in the liver as a reserve to be drawn upon after birth, but it is admitted that experiments were not sufficiently well controlled to justify any definite conclusion. The concentration of cobalt in the livers of newborn lambs from untreated ewes was greater than in the livers of the 6 months' old untreated lambs of the previous experiment, but the absolute amount of cobalt in the liver was the same despite the greater size of the livers in the older lambs. The significance of this was not understood. It has also been shown that topdressing pasture in Morton Mains with cobalt salts or limestone containing cobalt increases the cobalt content of the livers of lambs grazing on the pasture as well as preventing and curing sickness (Dixon and Kidson, 1940, 1941).

Composition of the Soil and Pasture

Soil. Chemical analyses of soils, including determinations of cobalt and copper, showed no significant difference between healthy and sick Southland soils, although the effectiveness of certain soil drenches appeared to be due to their high cobalt content (Dixon, 1936; Askew and Dixon, 1936). McNaught (1937) found that extraction of Morton Mains soils with decinormal hydrochloric acid gave values intermediate between those of healthy and bush-sick Rotorua soils, but there was no relationship between the cobalt content of the soil and the incidence of Morton Mains disease, the values for 4 affected areas ranging from 0·34 to 0·43 p.p.m. dry weight compared with samples from 2 healthy farms in Morton Mains which gave values of 0·26 and 0·29. This finding was confirmed by Kidson (1937), who extracted with concentrated hydrochloric acid samples of soil from areas in the Morton Mains district and found the cobalt content of 3 unhealthy soils to be 3·3, 4·0 and 4·8 p.p.m. compared with 2·8, 5·0 and 8·3, for healthy soils. Compared with values found for bush-sick soils, the unhealthy Morton Mains soils were fairly well supplied with cobalt.

Pasture. Dixon (Askew and Dixon, 1937 [3]), reporting the effect of topdressing Morton Mains pasture with cobalt, showed that in a control plot, upon which 8 out of 10 grazing lambs died of Morton Mains disease between the beginning of February and the end of May, the cobalt content of the pasture [illegible] p.p.m. D.M. ranged from 0·44 in October to 0·07 in May. It was suspected, however, that there had been contamination with soil. In experiments to test the effect of dressings of Southland limestone on the cobalt content of Morton Mains pasture and on the health of grazing lambs, Dixon and Kidson (1940) found that in two seasons in which no sickness occurred, the cobalt content of pasture from their control plots sampled from November to May ranged from 0·04 to 0·11 p.p.m. in December (oven dry basis), while in a third season they found that lambs on a control plot from December to April, where the cobalt content of the pasture varied from 0·04 p.p.m. D.M. in April to 0·07 in December, developed the symptoms typical for the area (Dixon and Kidson, 1941).

Askew and Dixon (1937 [3]) found that the application of 10 lb. cobalt chloride per acre was an effective means of overcoming Morton Mains sickness in grazing lambs. The cobalt content of the pasture, which before the application in October was 0·44 p.p.m. D.M., increased to 6·7 p.p.m. in November and thereafter decreased gradually until in May it had reached its former level. In pot experiments with Morton Mains soil Askew and Dixon (1937 [1]) found that superphosphate application favoured the uptake of cobalt by the pasture species grown in the pots. The simultaneous application of lime with cobalt chloride to the soil had a depressing effect on the uptake of cobalt by the pasture species, although the values obtained for the cobalt content of the plant material were much higher than for plants grown in untreated soil. Field experiments with mixed pasture at Marsden Research Farm, Nelson, indicated, however, that if the cobalt was not mixed with the limestone before application to the soil the depressing effect of the lime was not sufficiently serious to make the cobalt dressings ineffective (Askew and Dixon, 1937 [1]; Askew and Maunsell, 1937). Dixon and Kidson (1940) showed that a dressing of 3 tons limestone, supplying less than ¼ lb. cobalt sulphate per acre, markedly increased the cobalt content of Morton Mains pasture and of the livers of lambs grazing them, and in a season when Morton Mains sickness was prevalent, this treatment was also effective in preventing and curing the disease in weaned and unweaned lambs (Dixon and Kidson, 1941). They also compared the effect of topdressing with ¼ lb. cobalt sulphate alone and with 1 ton of ground limestone containing less than 1 p.p.m. of cobalt. One month after topdressing the cobalt contents of the pasture were 0·35 and 0·37 p.p.m. D.M., respectively. They concluded that limestones used for pasture topdressing on Morton Mains soils are beneficial and not detrimental.

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"MAIROA DOPINESS." NEW ZEALAND

Occurrence and Description of the Disease

Sheep grazing in the Mairoa district of the North Island of New Zealand have been reported to suffer from a disease known locally as "Mairoa Dopiness." The affected areas lie on the volcanic andesitic loams of the King country (Aston, 1928, 1932), and to a lesser extent on the more sandy Mangapohue type of Mairoa soil (Wright and Taylor, 1931).

No detailed description of the disease seems to be available but, according to Saunders (quoted by Askew and Rigg, 1932), the symptoms, including anaemia, are closely similar to those of sheep suffering from bush-sickness in the Kopaki area. Bones of "dopy" sheep are stated to be light (Aston, 1928), and Aston (1932) points out that "dopy" sheep differ from bush-sick sheep in this respect.

Experimental Studies

Feeding experiments. The disease was at first attributed to calcium starvation (Aston, 1928), mainly because of the lightness of the bones, the high lime requirement of the soil and because top-dressing pasture with lime had a beneficial effect, but no report of feeding tests with calcium has been found. Later, Askew and Rigg (1932) suggested that the disease might be due to iron deficiency which was then believed to be the cause of bush-sickness. Following on the discovery that bush-sick sheep recovered when fed cobalt supplements, McNaught (1939) pointed out that the excellent responses of sick animals to limonite and cobalt salt licks in the areas affected by the Mairoa disease indicated that the trouble was essentially of the same nature as the typical bush-sickness of the Rotorua, Taupo area, but no evidence is presented of controlled cobalt feeding experiments.

Composition of the blood. Saunders (quoted by Askew and Rigg, 1932) is reported to have found haemoglobin values of 40 to 45 per cent. (the scale is not stated) in the blood of sheep suffering from "dopiness" and bush-sickness compared with 60 per cent. in healthy controls. Analyses of the blood of affected and healthy sheep compared by Askew and Rigg (1932) are presented in Table 1.

TABLE 1

Composition of the Blood of Sick and Healthy Sheep

	Bush-sick Sheep (1) Glenhope	"Dopy" Sheep (1) Mairoa	Healthy Sheep (4) Nelson
Ca : mg per 100 g. blood	5·7	7·0	6·8
P : " " .	15·0	13·8	17·6
Fe : " " .	18·8	26·4	49·4
N : " " .	1675	1897	3207
Total solids : per cent.	11·3	12·5	20·2

Composition of bones. Young (1936) found that the bones of 9 "dopy" sheep were high in water content, ranging from 25·8 to 65·8 per cent. in raw bone compared with a maximum of 23·0 observed in healthy New Zealand sheep. The bones of "dopy" sheep also had a low ash : organic matter ratio, ranging from 1·29 to 1·40, expressed on dry fat-free bone, compared with 1·45 to 1·75 for healthy sheep. Values for a 3 year old and an 8 year old sheep from a bush-sick area were : water 37·8 and 15·3 per cent., ash : organic matter 1·48 and 1·81, respectively. The values for a "dopy" sheep whose health improved on pasture topdressed with lime and superphosphate were : water 17·7 per cent., ash : organic matter 1·75.

Composition of the Soil and Pasture

Soil. The high lime requirement of the soil in the Mairoa district was advanced as one reason for attributing Mairoa "dopiness" in sheep to calcium starvation (Aston, 1928), but Dixon (1931) was unable to show any significant difference between the lime requirement of soil in healthy and unhealthy areas of Mairoa. The fact that land after bush burning was highly productive but after some years deteriorated rapidly was taken as indicating leaching of calcium out of the soil (Aston, 1928). McNaught (1938) points out that this lag, before deterioration occurred, was not found in typical bush-sick areas where stock trouble developed immediately on pasture sown after bush burning. He considers, however, that this difference could be due to the ash from trees in the Mairoa country having a higher cobalt content than ash from trees in bush-sick areas, and gives analyses of the leaves of 2 common forest trees, Tawa and Mahoe, in support of this view. The cobalt stored up in the forest trees may have provided sufficient enrichment of Mairoa surface soil to permit the successful raising of cattle

and sheep for several years after bush burning. He suggests that, owing to the high rainfall, available cobalt has been leached out of Mairoa soil. Kidson (1937), however, gave the cobalt content of 2 samples of soil from the Mairoa ash shower as 4·5 and 5·3 p.p.m., and points out that similar values are found on the Tarawera ash shower which is healthy for stock.

Pasture. The pasture in the affected King country consists mainly of danthonia, yorkshire fog, and agrostis spp., clover and other leguminosae being almost entirely absent (Aston, 1928). An analysis of 21 year old unmanured Mairoa pasture as given by Aston (1928) is shown in Table 2.

TABLE 2

Composition of Mairoa Pasture

Ash	SiO_2	P_2O_5	CaO	MgO	Mn_3O_4	SO_3	Total N
11·59	3·44	0·66	0·83	0·51	0·037	0·82	2·54

The results are expressed as percentages of the sample dried to constant weight in the water oven. Analyses of pasture given by Young (1936) are presented in Table 3 (presumably per cent. D.M.).

TABLE 3

Composition of Mairoa and "Bush-sick" Pasture

	Ash	SiO_2	CaO	P_2O_5	N	Fe
Bush-sickness area						
(Kopaki). No fertiliser . . .	11·76	1·83	1·03	1·19	3·84	0·0064
Mairoa "dopy" area						
No fertiliser	7·81	1·12	0·99	0·41	3·06	0·009
Top dressed with lime and superphosphate	9·25	0·90	1·64	0·84	3·78	0·008

Data by McNaught (1938) show the average cobalt content of 10 samples of pasture from Mairoa "sick" areas to be 0·030 p.p.m., with a range of 0·020 to 0·044 p.p.m. D.M. The average cobalt content of 64 samples of pasture from typical bush-sick areas was 0·038 p.p.m., with a range of 0·009 to 0·079.

A seasonal variation in the cobalt content of Mairoa pasture is shown in the data of McNaught and Paul (1939), who analysed pasture samples from a farm in the Mairoa district where "dopiness" had been cured by means of cobalt licks (Table 4).

TABLE 4

Seasonal Variation in the Cobalt Content of Mairoa Pasture p.p.m. D.M.

	Jan.	Feb.	March	April	May	June	July	August	Oct.	Nov.	Dec.
1929	—	—	—	—	—	—	—	—	—	0·023	0·041
1930	0·027	0·032	0·031	—	0·028	0·048	—	0·026	0·031	0·019	0·023
1931	0·031	0·014	0·017	0·020	0·033	—	0·059	—	0·024	0·028	—

In King country, topdressing the "dopy" pasture with 5 cwt. limestone and 2 cwt. superphosphate per acre has proved beneficial to sheep grazing on it. Neither phosphate alone nor lime alone gave as good results as a combination of the two. This treatment effected a marked increase in clovers, and stimulated a vigorous growth of the English grasses (Aston, 1931; Wright and Taylor, 1931). Similar application to a reputed bush-sick area, as far as could be judged in an experimental period of 1 year, had beneficial effects on breeding ewes and their lambs (Wright and Taylor, 1931). Askew and Rigg (1932), favouring an iron deficiency theory for both bush-sickness and Mairoa "dopiness," suggested that the beneficial effect of the lime and superphosphate topdressing in Mairoa disease might be due to the increased clover content and consequent greater palatability of the grass, which is conducive to close grazing and relatively great contamination of the pasture with soil. The effect of topdressing with lime and superphosphate on the mineral content of Mairoa pasture is seen in Young's (1936) results given in Table 3; "dopy" sheep put on to such topdressed pasture improved in health, and the ash : organic matter of their bones was not below normal. Bonner *et al.* (1939) report a

top-dressing experiment in which treatment of pasture on the Mairoa andesitic shower with cobaltised superphosphate had a beneficial effect on ewes and lambs grazing on it ; control animals on pasture treated with ordinary superphosphate remained fairly healthy during the year, but did not show such good weight gains or wool yield. The pasture was dressed in the second week of February, and again in October. The effect on the cobalt content of the pasture is shown in Table 5.

TABLE 5

Effect of Fertilising on the Cobalt Content of Mairoa Pasture

Treatment	Cobalt : p.p.m. D.M.					
	23/11/37	22/3/38	27/4/38	29/7/38	21/11/38	28/2/39
Superphosphate	0·04	—	0·07	0·08	0·06	0·06
Cobaltised superphosphate (14 oz. Co sulphate per acre)	0·04	0·39	0·25	0·69	0·36	0·19

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ENZOOTIC MARASMUS. AUSTRALIA

Occurrence and Description of the Disease

This wasting disease occurs in sheep and cattle grazing the Denmark district near the south coast of West Australia. Holdings on which calves are known to have died from the disease are concentrated mainly in an area of approximately 5000 acres, the northern boundary of which lies 6 miles from the coast and the eastern boundary 3½ miles west of Denmark (Filmer, 1933). The disease has also been reported in the Nornalup, Walpole, Northcliffe and Redmond districts, and is probably present in other parts of the south-west (Filmer and Underwood, 1936). A similar condition has recently been reported in sheep in the south-eastern part of South Australia (McDonald, 1942). Filmer (1933) reported that a geological survey failed to show any difference between sound and unsound holdings in the Denmark district. The soil varied in colour from grey to red, and although in texture classified as a coarse sand, the general appearance was more of a sandy loam. Harvey (1937), in an investigation of Denmark soils, found that as several soil types generally occurred on any one holding it was difficult to correlate soil type and the incidence of the wasting disease. In general, however, the disease appeared to be associated with certain soil types, especially Wakundup gravelly sand, Kordapup sand, and an unnamed sandy loam, while certain other types never predominated on affected holdings. In South Australia only one soil type occurred on the affected property. It consisted of a layer of black friable clay overlying limestone (McDonald, 1942).

Enzootic marasmus is a wasting disease which affects cattle and sheep ; horses appear to be immune. Young cattle between 6 and 18 months are more susceptible than mature cattle, and sheep, lambs especially, are even more susceptible. The disease may occur at any time of the year, but is most common during the spring and early summer when green feed is abundant. It is characterised in both sheep and cattle by a gradually decreasing appetite until, finally, the animals lose condition and literally starve to death. This may occur in the presence of abundance of what appears to be excellent fodder. In cattle, lack of appetite for food is accompanied by a craving for such things as bark, sticks, bones, and earth. In cows lactation is depressed, abortion may occur, and it is difficult to get them in calf. Wasting in cows is often particularly rapid immediately after calving or abortion.

Cattle and sheep gradually become weaker, and if not treated die from 3 months to 2 years after the first appearance of symptoms. The course of the disease is more rapid in sheep and calves than in older cattle. Anaemia was said by Filmer (1933), and Filmer and Underwood (1936), to be characteristic of the disease. According to Filmer (1933), haemoglobin, red cell counts and colour indices are invariably low in affected animals, the anaemia being apparently hypochromic normocytic in lambs and hypochromic microcytic in calves. He found no evidence of excessive red cell destruction. More recently, however, Underwood and Beck (1941) have found that sheep which had been grazing for 9 months on "affected" pasture did not have low haemoglobin levels although they were emaciated and weak. McDonald (1942) points out that a striking feature in affected sheep in South Australia is the gross reduction in total blood volume. There is often heavy infestation with *Ostertagia*. *Haemonchus contortus* is rare, but infections with *Nemetodinis* sp., *Moniezia* sp. and *Chabertia ovina* are found. Haemosiderin can occasionally be demonstrated in the liver and invariably in large amounts in the spleen (Filmer, 1933).

According to Filmer (1933) it was common practice in the Denmark district to cure affected stock by transferring them to sound holdings.

Experimental Studies.

Feeding experiments. Because of the resemblance to bush-sickness, which was then believed to be due to a deficiency of iron, Filmer (1933) investigated the effect of treatment with iron compounds. It was found that the disease could be cured by massive doses of iron or by raw and dried liver, but there appeared to be no relation between the curative properties of iron compounds and their content of total iron or iron soluble in dilute acid. Later, Filmer and Underwood (1934), in studies of the curative action of limonite and of different fractions of a hydrochloric acid extract of it on heifer calves and sheep, showed that iron was not the curative element. An extract containing a negligible amount of iron was curative. Further fractionation showed that the curative element was in the fraction containing nickel, manganese, cobalt and zinc. Tests with supposedly pure nickel oxide suggested that nickel was the potent element, but subsequent work (Underwood and Filmer, 1935) indicated that the effectiveness of nickel salts was due to traces of cobalt, and this was confirmed by feeding tests in which affected sheep responded to treatment with pure cobalt chloride. The lowest daily dose of cobalt which induced optimum growth in sheep grazing on unsound farms in the Denmark district was found to be 0·1 mg. cobalt in the form of cobalt chloride (Filmer and Underwood, 1937). Cattle were cured and maintained in good health for periods up to 1 year by the administration of cobalt chloride at the rate of 0·3 to 1·0 mg. cobalt daily. With a suboptimal dose of 0·05 mg. cobalt the addition of 0·02 mg. nickel was found to increase the effect of the cobalt. Of the various supplements which had proved effective in curing or preventing enzootic marasmus all except liver were found to contain significant quantities of cobalt. It was suggested that the potency of liver might be due to the presence of some stored factor, and that cobalt may function by helping to produce this factor in the body. Filmer and Underwood (1936) reported that 2 affected cows given daily doses of cobalt showed an increase in milk production above the general increase caused by seasonal improvement in the pasture.

The possible significance of copper in the etiology of the disease was investigated by Underwood and Beck (1941). No additional improvement in the condition of affected sheep treated with cobalt together with copper occurred in comparison with those treated with cobalt alone, although the latter group received no copper other than that provided by the pasture for a period of 22 months. Further, no stringy wool was observed in sheep, and stringy wool is considered highly characteristic of copper deficient areas (see p. 50), and is said to develop in male and female sheep within a few months of depasturing on copper deficient grazing. It was concluded that enzootic marasmus is due to a deficiency of cobalt uncomplicated by lack of copper. In South Australia McDonald (1942) found that mineral mixtures and cobalt alone were beneficial, but anthelmintic treatment was without effect.

Composition of the blood. Values as low as 6·5 and 4·5 g. haemoglobin per 100 c.c. for a calf and a hogget and 2·8 and 3·7 million red cells for a cow and a lamb have been reported in affected areas (Filmer, 1933). Filmer and Underwood (1937) found rises in haemoglobin and red cell count in response to cobalt treatment in sheep whose condition, as judged by weight change, was improving. These rises were succeeded by remarkable falls followed by further rises in many of the experiments. Similar falls were observed in heifers recovering on a sound farm. Underwood and Beck (1941) found that sheep which had been grazing on affected pasture for 9 months and were showing the emaciation and weakness characteristic of the disease did not have haemoglobin levels which could be regarded as low. Some showed lower levels after 1 month's cobalt treatment, but by the end of the third month all the values were high. Although the haemoglobin levels fluctuated throughout treatment, at no

time were values found indicative of even the mildest anaemia. There was no suggestion of lower haemoglobin values in sheep receiving cobalt alone compared with those receiving both cobalt and copper, although the former had subsisted for 22 months without a copper supplement. Significant falls in the copper content of the blood in this group were observed during the last 6 to 7 months of treatment.

Composition of organs. Underwood (1934) found a high iron content in liver, spleen and kidney from 9 young sheep and 2 calves dying from enzootic marasmus as compared with normal controls. Larger masses of haemosiderin were present in the affected spleens. Since Filmer (1933) had previously found no evidence of excessive red cell destruction, Underwood suggested that the high concentrations of iron in spleen and liver are due to the absence from the food supply of some factor necessary for its utilisation. Underwood and Beck (1941) found normal iron contents of the liver, spleen and kidney of affected sheep treated with cobalt for a period of 3 months. Underwood and Harvey (1938) investigated the cobalt status of the livers of 10 affected sheep and found that with one exception all had a cobalt content of 0·07 p.p.m. D.M. or less, compared with the livers of 10 healthy sheep which, with one exception, contained 0·20 p.p.m. cobalt or more. Underwood and Beck (1941) found that the copper content of the livers of sheep cured of enzootic marasmus by cobalt treatment were well below the normal levels usually reported for healthy sheep, though appreciably higher than the values reported from areas known to be highly deficient in copper.

Composition of the Soil and Pasture

Soil. Harvey (1937) analysed Denmark soils for cobalt and Underwood and Harvey (1938) considered the data in relation to the incidence of enzootic marasmus. They found that the cobalt content of " affected " soils ranged from 0·1 to 1·5 with a mean of 0·6 p.p.m. on dry basis. Values for adjacent healthy areas were 0·2 to 32·0 with a mean of 5·6, and for healthy soils outside the district, 0·5 to 40·0, with a mean of 10·4 p.p.m. The cobalt content of non-agricultural soils ranged from 0·1 to 0·5 with a mean of 0·3. In general, the cobalt content of " affected " soils was much lower than of healthy soils, but some healthy soils also gave low values.

Pasture. Virgin country in the Denmark area, on account of the heavy timber and undergrowth, carries little or no natural pasture. Timber has been cleared away for the artificial establishment of permanent pasture which consists mainly of *Trifolium subterraneum, T. minus, T. repens, Lotus major, Paspalum dilatatum, Holcus lanatus, Festuca* spp., and *Bromus hordaceus.* The most common tree on affected holdings was karri (*Eucalyptus diversicolor*), and the disease has never been known on land on which jarrah (*E. marginata*) had been the predominant timber (Filmer, 1933).

Data have been presented by Harvey (1937) and Underwood and Harvey (1938) for the cobalt content of 12 samples of Denmark pasture consisting mainly of *Trifolium subterraneum.* The cobalt content of pasture from affected areas ranged from 0·02 to 0·07 with a mean of 0·04 p.p.m. D.M., and those of healthy adjacent pastures ranged from 0·04 to 0·30 with a mean of 0·13. In 6 samples from healthy areas outside the affected district, the cobalt content ranged from 0·03 to 0·43 with a mean of 0·18 p.p.m. D.M. Although all the affected pastures were considerably lower in cobalt than most of the healthy pastures, several of the pastures which had induced no evidence of enzootic marasmus in grazing stock also showed low values.

The possible significance of copper in the etiology of enzootic maramus was investigated by Underwood and Beck (1941). They found that the copper content of pasture (principally subterranean clover) on Wakundup soil (the predominant type on affected holdings) ranged from 2·2 to 6·5 with a mean of 3·7 p.p.m. D.M., while that on Scotsdale gravelly loam or alluvial soil (the predominant types on holdings unaffected with the disease) ranged from 4·3 to 22·2 p.p.m. copper with a mean of 9·9. They considered that the average copper content of the affected pastures was marginal for dry sheep and cattle and probably too low for breeding sheep, but feeding experiments and analyses of sheep organs (see above) gave no indication that copper deficiency was concerned in the etiology of enzootic marasmus.

Underwood and Harvey (1938) pointed out the need for data on seasonal variations in the cobalt content of pasture, but no study has yet been published for areas with enzootic marasmus. They also indicated the need for data to show if low values for soil cobalt necessarily imply correspondingly low values for the pasture in these areas.

Underwood and Harvey (1938) found that the cobalt content of affected pastures was increased from 0·08 p.p.m. to 0·22 and 0·96 p.p.m. D.M., respectively, in September, 4½ months after treatment with cobalt acetate at levels of 0·5 lb. (54 g. cobalt) and 10 lb. (1070 g. cobalt) per acre. In December, 7½ months after fertilising, the values were 0·30 and 1·60 p.p.m., respectively.

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"NUTRITIONAL ANAEMIA" OF SHEEP. NEW SOUTH WALES

Occurrence and Description of the Disease

"Nutritional anaemia" in sheep has been reported in the New England district of New South Wales. The country most severely affected is on a granite formation. The condition, known locally as "poorness of blood," is characterised by gradual loss of condition, pining, and weakness leading to death. The blood is said to be thin and watery, skin and mucosa are pale, and a typical diseased sheep in May showed haemoglobin 46 per cent. (method not given), red cell volume 23 per cent. and red cell count 8 million per c.mm. According to general opinion loss of condition begins early in autumn and the disease is essentially a winter complaint. Animals are heavily parasitised. All sheep irrespective of age and breed are affected. Sheep removed to other districts recover.

Experimental Studies

Feeding experiments. Anthelmintic treatment, together with the feeding of a salt lick containing iron or a supplement of dicalcium phosphate, had little effect on the incidence of the anaemia. The experimental sheep which did not die were then transferred to good pasture or given a diet of oats and lucerne. They recovered from the anaemia and gained in weight. Results are given for the blood picture of 1 sheep given wider grazing range, drenched with cobalt sulphate and given iron ammonium citrate daily. Haemoglobin rose from 46 to 70 per cent. (method not given), red cell volume from 23 to 33 per cent., and red cell count from 8 to 10·2 million per c.mm.

Composition of the Pasture

Analyses of 2 samples of pasture showed that they contained 0·322 and 0·409 per cent. CaO, 0·194 and 0·261 per cent. P_2O_5, 0·924, and 1·149 per cent. nitrogen and less than 4 p.p.m. copper on a dry matter basis.

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NAKURUITIS. KENYA

Occurrence and Description of Disease

The disease occurs in grazing cattle in the Nakuru district of Kenya from which its name is derived. It appears in animals pastured for 4 to 6 months in the area, and is characterised by emaciation; anaemia is said to be marked, and in advanced stages there is loss of power and co-ordination of the limbs. *Post mortem* examination showed only a typical picture of inanition. Pigmentation of the splenic pulp was also noted. The disease has a seasonal incidence, and young cattle are said to be more susceptible than older animals. Affected cattle exhibit a craving for certain local edible earths, and the native custom of giving cattle access to such licks helped to keep down the incidence of the disease. Removal to an area where stock remained thrifty also proved beneficial provided the animals were not too badly affected.

Experimental Studies

Feeding experiments. Because of the resemblance to bush-sickness, mineral mixtures high in iron were tried with beneficial results (Orr, 1929). Affected animals showed a distinct craving for such beneficial mixtures (Econ. Advisory Council, 1931) and local edible earths which are licked by cattle were found to contain a high percentage of iron oxide (Gunn, 1931). Nakuruitis was very severe in 1936, and treatment with iron oxide was not giving the results expected, so cobalt treatment, which had proved beneficial in controlling enzootic marasmus, was tried in Kenya. Preliminary reports indicated that cobalt had a beneficial effect on the weight gain and blood picture of affected animals (Dept. Agric., 1936; Vet. Dept., 1937).

Composition of the Pasture

Analysis of 31 samples of Nakuru pastures showed the following percentage values on D.M. basis (Econ. Advisory Council, 1931):

	N	CaO	P_2O_5	Na_2O	K_2O	Cl
Range . .	0·317–1·952	0·277–0·684	0·047–0·626	trace–0·405	0·333–3·470	0·100–0·840
Mean . .	0·888	0·412	0·305	0·064	1·586	0·332

No analysis for cobalt content appears to have been published.

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PINING. UNITED KINGDOM

Occurrence and Description of the Disease

A wasting disease known as pine or pining is widespread among sheep in Great Britain. It occurs on pastures in Orkney, in the Hebrides, and on the coasts of Argyllshire. The majority of Scottish counties are affected, especially the border counties, and on the English side of the border pining areas are found in Northumberland. The disease has also been reported on Dartmoor and occurs in certain districts in North Wales. Pining in cattle has been reported in Tiree and in Easter Ross, Scotland. McGowan and Smith (1922) found that pining was not limited to any special type of rock or soil. Although the majority of farms in the Cheviot area are situated on or near the porphyrite formation, all of these are not pining farms, and affected areas, which extend west from Selkirk and Peebles into Galloway, are over the Silurian rocks. Later, Corner (1939), in a study of affected areas in the Scottish Cheviot region, observed that pining farms are mainly situated on the Andesite formation, a hard, volcanic type of rock which usually produces a shallow soil of coarse texture. Greig *et al.* (1933), investigating the condition on the Island of Tiree, found that the affected areas consist of blown sand containing a high proportion of shell fragments, while the healthy areas are on the gravel or gravelly sand of raised beaches. According to Corner (1939) affected areas in Orkney and on the Argyllshire coasts have the same type of soil heavily impregnated with shell sand as Tiree pining areas. Patterson (1938) found that Dartmoor pining areas are on granite, and healthy change pastures are on Devonian conglomerates or Middle Devonian shales.

Pining in sheep and cattle is said to be a condition of emaciation and anaemia resembling bush-sickness in New Zealand or nakuruitis in Kenya. In young animals growth is greatly retarded. Loss of appetite is mentioned by Corner (1939). Anaemia has been observed in some but not in all areas. According to Patterson (1938), who described the disease as it occurs on Dartmoor, Welsh sheep are susceptible while Shetland sheep appear to be immune. Corner (1939), however, states that while lambs are more susceptible than older sheep, all ages and all breeds in common use are liable to develop the disease. In North Wales pining occurs in lambs but apparently not in ewes or cattle. Lambs not only cease to thrive but frequently die (Rowlands, 1940). The critical period appears to be between March and August. McGowan and Smith (1922) stated that on the average pining is more prevalent in a wet than in a dry year, and attributed this to the fact that in a dry year sheep persist

in grazing the shorter herbage of the hilltops, but when that becomes dry and short they are obliged to frequent the coarser herbage of the lower ground to some extent. Rowlands and Harbour (1941), however, find that pining in sheep on permanent pasture in North Wales is more severe in dry seasons than in wet. Severe parasitic infestation is often observed in affected sheep. (Stewart, W. L. and Piercy, 1935; Stewart, W. L. and Ponsford, 1936, 1940; Patterson, 1938; Greig *et al.*, 1938; Rowlands, 1940; Rowlands and Harbour, 1941.)

Methods of management which are beneficial on pining farms have been described by McGowan and Smith (1922). Change to healthy pasture is curative; keeping the sheep moving so that they get a more varied type of diet is also beneficial; sheep can be kept on pining farms all the year round without change of pasturage provided they get a regular ration of linseed cake, and putting the sheep to feed on a field of turnips is also beneficial. Greig *et al.* (1933) found that alternating the animals at monthly intervals between healthy and pining land was protective against pine in Tiree, and Patterson (1937) reported similar experience with Dartmoor pining.

Experimental Studies

Feeding Experiments. Beneficial effects, preventive and curative, followed the treatment with iron compounds of cattle in Tiree and Easter Ross (Greig *et al.*, 1933; Brown, 1935) and of lambs on the Scottish borders (Greig *et al.*, 1938), but improved condition was not always accompanied by a rise in haemoglobin (Greig *et al.*, 1933, 1938). Greig *et al.* (1938) concluded that the beneficial factor in the crude iron compounds was not copper, since copper therapy was without effect. Patterson (1938) found that a mineral supplement containing cobalt, manganese and zinc gave an initial advantage in liveweight gain to affected lambs on Dartmoor compared with untreated controls. This advantage was not maintained, but at the end of 2 months the treated lambs were in better condition, although of the same liveweight as untreated lambs. Later, Corner and Smith (1938) showed that 14 daily doses of 1 mg. cobalt per head over 3 weeks cured the disease, or prevented its appearance for 6 months, in sheep newly brought on to pining land on the Scottish borders. Attempts to cure the disease with 0·5 mg. doses of cobalt were successful only where pining was in its early stages. In a further report Corner (1939) states that cobalt treatment for preventive purposes has been adopted on 30 pining farms in Scotland with uniformly successful results. On some farms the incidence of the disease has been reduced from 8 or 9 per cent. to nil. The experimental results of Corner and Smith (1938) have been criticised by Stewart, W. L. and Ponsford (1940) on several grounds. They pointed out that the sheep, said to be pining after only 6 weeks on pining land, were treated with cobalt without controls to show that the improvement would not have taken place in any case, and further that the sheep which were deemed to be recovering showed an average liveweight gain of 3·2 lb., which was not much different from the average liveweight gain of 2·6 lb. which had, before the treatment, been taken as indicative of pining. At this stage Stewart, W. L. and Ponsford's own experiments showed that no beneficial effect, as judged by liveweight and haematocrit values, was obtained when sheep maintained for 2½ years on pining land were treated with 3 mg. cobalt daily for 5 months, more sheep dying of pining in the treated than the untreated group. In reply, Corner and Smith (1940) pointed out that, under the conditions of Stewart and Ponsford's experiments, the treated animals may have been permanently injured before treatment commenced. The main part of these experiments showed that, over a period of 3 years, either anthelmintic treatment or treatment with a mineralised supplement providing, in addition to extra calories and protein, 1·96 g. sodium, 3·03 g. chlorine, 2·19 g. iron, 6·80 g. calcium, 3·51 g. phosphorus, 0·49 mg. manganese, 2·67 mg. cobalt, 0·70 mg. potassium, 2·30 mg. iodine and 7·59 mg. copper daily, reduced the incidence of pining, as compared with control groups, and a combination of the two treatments gave the best results as judged by liveweight increase and haematocrit value.

Emphasis had earlier been laid by Stewart, W. L. and Piercy (1935) on parasitic infestation as a possible cause of pining when they showed that the faecal worm egg count varied with the severity of the disease and the intensity of the anaemia; differential leucocyte counts showed a picture similar to that recorded in well-marked infestations with gastro-intestinal parasites, and *post-mortem* examination of 13 pining sheep gave evidence of parasitic gastritis in all cases. The number of parasites was greatest during the summer months when pining was at its height; in cases which survived until the winter there was a reduced total parasite count. The anaemia was not identical with that of haemonchosis. Later, Stewart and Ponsford (1936), although they considered the main symptoms of acute pining to be due to infestation with nematode parasites, pointed out that in the chronic stage of the disease the number of parasites was insufficient to account for the degree of anaemia and emaciation found. They concluded that Border pining in sheep was due to the combined effects of malnutrition and gastro-intestinal parasitic infestation. Following the work of Corner and Smith in 1938, and despite their own failure to cure the condition with cobalt, they admitted (1940) the possibility that cobalt deficiency might decrease initial resistance to worm infestation. On the other

hand, Patterson and Corner and Smith on general grounds, and because of their success with cobalt treatment, considered cobalt deficiency rather than parasitic infestation as the prime factor in the pining which occurs on Dartmoor and in Scotland. The issue has been clarified by the work of Rowlands. Investigating pining in lambs on permanent pasture in North Wales, Rowlands (1940) found no relationship between total worm burden or differential species count and liveweight gain. Anthelmintic treatment with copper-nicotine sulphate had a beneficial effect on weight gain, but the worm counts in treated and untreated sheep were almost the same. Later, Rowlands and Harbour (1941) suggested that the beneficial effect may have been due to contamination of the copper-nicotine sulphate with cobalt. In experiments at 3 centres it was found that pining in sheep was in one case prevented equally by phenothiazine as anthelmintic and by a mixture of trace elements; in another case only the phenothiazine was effective, and at the third centre the phenothiazine was ineffective while the trace element mixture was successful. Further experiments at this centre indicated that cobalt was the potent element in the mixture. Worm counts showed that the phenothiazine was an efficient anthelmintic. Concurrently with this work Stewart, J. *et al.* (1941) found that pining in sheep in Ross-shire could be cured by cobalt administration but agree that pining may be due to different primary causes in different areas.

Composition of the blood. In an investigation of pining in Tiree Greig *et al.* (1933) found haemoglobin values of 6·28 to 8·70 g. per 100 c.c. in the blood of cattle said to be pining compared with 7·[illegible] to 10·75 g. in the blood of cattle whose condition was fair to good.

Stewart, W. L. and Piercy (1935) examined the blood of pining and non-pining sheep on an affected border farm at intervals over a period of 18 months. The low values obtained between June and August and the general improvement which was observed by December of the same year are shown in Table 1.

TABLE 1

Blood Picture of Pining and Non-pining Sheep

	Cell Volume per cent.	Mean Cell Volume c.μ.	R.B.C. Mill. per c.mm.	Hb g. per 100 c.c.	Colour Index	White Blood Cells per c.mm.
June						
5 pining sheep	17	29·5	5·77	6·12	1·06	4,740
5 non-pining sheep	30	28·7	10·52	11·43	1·10	7,180
December (except 1 which was Oct.)						
4 pining sheep	30	29·2	10·48	9·25	0·89	8,750
5 non-pining sheep	33	28·1	11·72	10·16	0·87	10,960

This improvement was not maintained after December. Similar results were obtained on another pining farm. Greig *et al.* (1938) found that top quality lambs put on pining Border pasture in Augu[illegible] were pining by December. The haemoglobin estimated in November and December ranged from 7·2 to 11·5 g. per 100 c.c., with an average of 9·4 g. No morphological abnormality of the red cells was observed. Stewart, W. L. and Ponsford (1940) reported a reduction in haematocrit values in 2 groups of 16 lambs kept on pining pasture for 2½ years from 28·3 and 29·0 (Sept.) to 25·0 and 23·0 (March); corresponding reductions for 2 groups of 16 ewes were from 28·5 and 30·4 (Sept.) to 24·0 and 23·0 (March). Rowlands and Harbour (1941) were unable to demonstrate any abnormality in the blood of affected lambs.

Eden (1939), in a study of the copper content of the blood of sheep, found no relationship between that of Northumbrian sheep and the incidence of pining.

Composition of the Soil and Pasture

Soil. Analysis of pining and non-pining soils in Tiree by Greig *et al.* (1933) showed that the soil of affected areas was low in available iron as measured by solubility in 5 per cent. oxalic acid. This finding was in harmony with the curative and preventive effect of crude ferric oxide on sheep, and it was concluded that pine was probably due to a deficiency of iron in the pasture and/or of available iron in the soil. It was thought that the unusually high content of calcium carbonate in the 'affected' soil might limit the amount of soil iron available directly to the animal. Since Orr (1929) had pointed out the resemblance between pining in Scotland and bush-sickness in New Zealand, Patterson (1937) was led by the results of Askew's work on bush-sickness to suspect a deficiency of cobalt as the cause of pining in Dartmoor sheep. He analysed moorland soils on which sheep suffered from pining and

found a mean cobalt content of 3·9 p.p.m. compared with 16·7 p.p.m. in lowland soils on which sheep recovered. Patterson later (1938) reported that between healthy and pining soils in Devon there was no significant difference in content of available iron but the average cobalt content of sick soils was 4 p.p.m. compared with 19·6 p.p.m. in healthy soils. The cobalt content appeared to be related to the magnesium content of the parent rock. Corner and Smith (1938) investigated pining and non-pining soils from the Cheviot region of Scotland. Soils were extracted with an ammonium oxalate-oxalic acid solution of *p*H 3·2 to 3·3 and analysed for iron, copper, manganese and cobalt. The results did not indicate a relationship between pine and the iron, copper or manganese content of the soil. The cobalt contents of all pining soils expressed as p.p.m. by volume were under 2 for both spring and summer, while in the non-pining soils the cobalt content ranged from 2·8 to 5·4 with the exception of one low summer value of 0·8 for a non-pining farm on peat which had, however, a spring value of 2·8. Stewart, J. *et al.* (1941) carried out a systematic spectrographic investigation of soils from pining farms in Ross-shire, Inverness-shire and Sutherlandshire over a period of 3 years. They found that most of the pining soils contained less than 5 p.p.m. of cobalt while the range for all soils examined spectrographically at their institute was 1 to 300 p.p.m. with a mode of about 10 p.p.m. In experimental plots of soil containing 2·9 p.p.m. of cobalt it was found that topdressing with cobaltised fertilisers increased the cobalt content of the soil in proportion to the amount of cobalt applied up to a value of 9·6 p.p.m. 10 weeks after an application of 80 lb. cobalt chloride per acre. The readily soluble cobalt or the amount soluble in dilute acetic acid accounted for about one-tenth of the total soil cobalt. Analyses of readily soluble cobalt in the soil in the following year showed that even after 15 months the application of 80 lb. cobalt chloride had a considerable residual effect (Stewart, J. *et al.*, 1942).

Pasture. There does not appear to be any reliable evidence in the literature relating pining to the type of herbage, but when flocks are changed to non-pining land to prevent the onset of the disease preference is given to heather or moss (Corner, 1939). Godden and Grimmett (1928) reported that, in general, the iron content of pasture was lower in pining than in non-pining areas. Greig *et al.* (1933) analysed samples of grass from pine and non-pine areas of Tiree. They found that the former were poorer in nitrogen and phosphorus but richer in lime than the latter. Iron was not estimated owing to contamination of samples with soil. Analyses of pasture from farms in Northumberland where pining occurs showed a low content of lime, phosphorus and iron (Stewart, W. L. and Ponsford, 1936) and a copper content ranging from 7 to 10 p.p.m. D.M. (Eden, 1939). At this stage attention was directed by Patterson (1937) to the possibility of pining being due to a cobalt deficiency in the pasture. He showed that pasture on sick areas on Dartmoor had a mean cobalt content of 0·20 p.p.m. compared with 0·45 p.p.m. cobalt in pasture from recovery areas. Corner and Smith (1938) in their analyses of spring and summer herbage from pining and non-pining areas of the Scottish Cheviot district found no relation between the disease and the iron, copper or manganese in the pasture. Spring values for cobalt ranged from less than 0·05 to 0·20 p.p.m. and summer values from less than 0·05 to 0·12 p.p.m. and did not appear to be related to the incidence of the disease. Administration of cobalt, however, prevented the occurrence of the disease in sheep on pining land, and it was pointed out that, since the cobalt content of New Zealand pasture had been shown by Askew and Maunsell to fall during the growing season, individual results for the cobalt content of pasture might be liable to misinterpretation. Rowlands and Harbour (1941) stated that in the pining areas of North Wales there was no indication of general malnutrition, a view said to be confirmed by pasture analyses, but no analytical data were presented.

In a farm in Easter Ross where sheep suffer from pining, Stewart, J. *et al.* (1941, 1942) obtained an increased cobalt content of pasture with increased applications of cobalt chloride up to a value of 8·17 p.p.m. obtained 10 weeks after an application of 80 lb. cobalt chloride per acre when the values for untreated controls were 0·11 to 0·20 p.p.m. There was still a considerable residual effect 15 months after the application. Grazing experiments with lambs showed that pining was prevented on parts of a field treated with 2 lb. cobalt chloride per acre while lambs on untreated parts of the same field began to pine. The cobalt contents of the pasture on control plots in July before the lambs were put on to graze were 0·09 and 0·08 p.p.m.

Mitchell *et al.* (1941) drew attention to the high molybdenum content of pasture upon which a form of pining in cattle not definitely attributed to cobalt deficiency had been reported. This pasture (A) contained 6·3 p.p.m. of molybdenum and 0·07 p.p.m. D.M. of cobalt compared with 1·7 p.p.m. of molybdenum and 0·08 p.p.m. D.M. of cobalt in a sample of untreated pasture (B) upon which pining in sheep could be cured and prevented by application of 2 lb. cobalt chloride per acre. It was suggested that in the former case the pining may have been due to a combined deficiency of cobalt and excess of molybdenum in the pasture. Application of 2 lb. cobalt chloride per acre did not raise the molybdenum content of the latter pasture above 2·2 p.p.m., but the other pasture showed an increase to 9·2 p.p.m. (Table 2), at which level it was said to be approaching " teartness." The molybdenum of " teart " pasture in Somerset is, however, usually much higher than this (see p. 53).

TABLE 2

THE CO AND MO CONTENT OF "PINING" PASTURES (P.P.M. D.M.) 15 MONTHS AFTER FERTILISING WITH CO.

$CoCl_2\ 6H_2O$ applied lb. per acre	Pasture A		Pasture B	
	Co	Mo	Co	Mo
0	0·07	6·3	0·08	1·7
2	0·20	9·2	0·22	2·2
10	0·89	10·0	0·63	2·4
80	2·75	14·2	3·20	7·5

Mitchell *et al.* suggest, in view of these results, that caution must be exercised in the general use of cobalt-rich fertilisers, and since it has been shown that the uptake of molybdenum increases with decreasing soil acidity, the use of cobalt-rich lime as has been suggested in New Zealand may be dangerous on pasture of type A.

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"GRAND TRAVERSE" DISEASE. MICHIGAN, U.S.A.

OCCURRENCE AND DESCRIPTION OF THE DISEASE

For many years this disease of cattle has been known in several regions in Michigan, commonly on light sandy soils, but occasionally on heavier soils. The symptoms are severe emaciation, staring coat, loss of appetite, refusal to drink and pica. There is marked anaemia, severely emaciated animals showed haemoglobin values ranging from 6 to 8 g. per 100 c.c. compared with 8 to 10 g. for mildly affected, and an average of 13 g. for an unaffected college herd. Blood analyses revealed no evidence of ketosis or phosphorus deficiency. Improvements in feeding or moving cattle to other sources of feed usually results in a cure.

EXPERIMENTAL STUDIES

Feeding experiments. Experimental addition of cobalt to the winter rations of cattle which had been affected for several months resulted in a spectacular restoration of appetite in 3 to 10 days, with marked improvement in condition and in milk production of lactating cows. After administration of cobalt there was a decrease of 10 to 20 per cent. in the haemoglobin level followed by a gradual climb towards normal. It is suggested that the haemoglobin drop was apparent rather than real, and was probably due to an increase in the volume of the blood following the great increase in the intake of feed and water ; later the haemoglobin increase gained on the blood volume and a rise in haemoglobin level was shown.

COMPOSITION OF HAY

Hay from affected areas contained 0·03 to 0·06 p.p.m. of cobalt, compared with 0·12 p.p.m. from a healthy area, which seemed to confirm the indications of the feeding experiment that the disease was due to deficiency of cobalt in the feed (Killham, 1941).

Reference : Killham, B. J. (1941). *J. Amer. Vet. Med. Assoc.*, **99,** 279.

"NECK-AIL." MASSACHUSETTS, U.S.A.

A disease resembling the bush-sickness of New Zealand has been reported in grazing cattle in Massachusetts (Archibald *et al.*, 1938). The condition responds to drenching with iron ammonium citrate. The authors refer to the work in Australia and New Zealand, which indicates that this type of nutritional anaemia is due to cobalt deficiency, but no similar work appears to have been done in Massachusetts.

Reference : Archibald, J. G., Kucinski, K. J., Brooke, R. O. and Freeman, S. L. (1938). *J. Dairy Sci.*, **21**, 59.

COBALT DEFICIENCY. CANADA

Although there is no direct evidence of a naturally occurring cobalt deficiency in Canada, the condition has been produced experimentally in sheep confined for long periods to a ration consisting chiefly of non-leguminous hays grown in the Province of Alberta (Bowstead and Sackville, 1939 ; Bowstead *et al.*, 1942 [1]).

Ewes fed a ration of hay, half of which was oat hay and the other half timothy, brome grass or [illegible]irie hay, with ground oats, iodised salt and monocalcium phosphate, showed loss of appetite and [illegible]lessness, followed by loss of weight, unthriftiness and death. Affected ewes produced small, weak lambs, insufficient milk to nourish the lambs, and fleeces weak in fibre. The haemoglobin, red cell count and packed cell volume of 5 affected ewes averaged 9·2 g. per 100 c.c., 9·7 million per c.mm. and 23·8 per cent., respectively, compared with averages of 12·8 g. per 100 c.c., 13·3 million per c.mm. and 41·6 per cent., respectively, for 2 healthy ewes.

Some benefit was obtained by supplementing the ration with various concentrates, with calcium and phosphorus and by giving the sheep access to pasture, but the most striking result was obtained with alfalfa hay or its ash. Iron and copper supplements were detrimental rather than beneficial, and attention was directed to cobalt as the result of the work of Neal and Ahmann in Florida and Askew and Dixon in New Zealand (Bowstead and Sackville, 1939). It was found that addition of 4 mg. cobalt twice weekly to the ration prevented the occurrence of the symptoms shown by 28 out of 31 control ewes receiving no cobalt supplement. A group of 8 of the control ewes which were showing symptoms were then selected for treatment with cobalt and made rapid gains in weight (Bowstead *et al.*, 1942 [1]). Subsequent experiments showed that cobalt in the form of a cobaltised salt mixture or as a solution of cobalt chloride were equally beneficial (Bowstead *et al.*, 1942 [2]).

Estimations of cobalt in blood, liver and spleen were made on a few thrifty and unthrifty animals from the control group. The values for blood were not significantly different, but the concentrations of cobalt in the liver and spleen of the unthrifty animals were much reduced and the former were similar to values observed by Askew and Dixon for the livers of affected animals in the bush-sickness investigations in New Zealand (Bowstead *et al.*, 1942 [1]).

The average cobalt content of samples of feeds used in the experiment are shown in the following table :—

	No. of Samples	Co : p.p.m.
Oat hay . . .	2	0·02
Timothy hay . .	5	0·01
Oat chop . .	2	0·02
Slough hay . .	1	0·015

Similar low average values were obtained for samples of oat and timothy hay collected from other parts of the Province (Bowstead *et al.*, 1942 [1]). The alfalfa hay which, in the 1939 experiment, gave such good results when fed as a supplement, contained 0·16 p.p.m. of cobalt, although grown on the same soil as the non-leguminous hays. Samples of alfalfa hay from the Lethbridge area of Alberta contained only 0·012 p.p.m. of cobalt, and it is suggested that sheep in that part of Alberta may be receiving insufficient cobalt for the maintenance of normal growth and thrift (Bowstead and Sackville, 1939).

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CHAPTER II

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO DUAL EFFICIENCY OF COBALT AND COPPER

INTRODUCTION

This chapter consists of an account of the investigations of coast disease in Australia and " salt sick " in Florida. The evidence that cobalt and copper are involved in the etiology of these diseases is discussed in Chapters I and III.

COAST DISEASE. AUSTRALIA

Occurrence and Description of the Disease

A survey of the distribution of coast disease, a wasting disease affecting grazing animals in the state of South Australia (Marston *et al.*, 1938) showed that large areas of coastal mainland, mostly occupying a westerly or southerly aspect, a large area of Kangaroo Island and probably the whole of Boston and Thistle Islands were affected. On a conservative estimate, more than 2000 sq. miles were involved. The disease has also been reported on King Island, Tasmania (Philp, 1933), and in Western Australia, but investigations of coast disease in the latter state have been confined to a few small areas on the west and south coasts, notably on the west coast, west of Gingin ; no survey has been made of affected country (Bennetts, 1940). The disease appears to occur mainly on highly calcareous coastal dunes, the soil of which is composed almost entirely of wind-borne shell fragments, but it has recently been pointed out that in other areas of South Australia, which are geologically unrelated to the shell sands, similar conditions prevail and limit the well-being of sheep. These areas are not yet completely mapped (*J. Dept. Agric. S. Austral.*, 1941).

The resemblance between coast disease, enzootic marasmus, bush-sickness, pining and nakuruitis has been pointed out by Marston *et al.* (1938). The principal symptoms of the disease are lethargy, weakness, loss of appetite, emaciation and blanching of the visible mucous membranes. Progressive anaemia is associated with the emaciation, but sheep may show recognisable symptoms of coast disease prior to the appearance of obvious signs of anaemia. During the progress of the disease, the haemoglobin levels fall. This change is said to be accompanied by a decrease in blood volume and reduced efficiency of the heart.

Autopsy reveals signs of general malnutrition, oedema, atrophy of the viscera, lightness and fragility of bones. A distinctive feature of the disease is intense haemosiderosis of the liver, pancreas and spleen. Sheep, cattle and goats are known to be affected, but horses are apparently immune, although according to Philp (1933) the rearing of foals on certain " coasty " areas of King Island is said to be impossible. On badly " coasty " areas young cattle, adult wethers and dry ewes as well as weaners and pregnant and lactating ewes develop the disease within a few months. On " coasty " areas weaners rapidly decline and lactating or pregnant ewes, dry ewes and wethers become affected in that order. On semi-" coasty " areas weaners fail to thrive and there are some losses, while adult animals are not noticeably affected. Sick animals brought there, however, fail to recover. Removal of sick animals to " sound " country is followed by rapid improvement in health.

Philp *et al.* (1938–39) report that an investigation of " coasty " sheep in King Island showed no significant degree of parasitism.

Experimental Studies

Feeding Experiments. A series of experiments with sheep on the " coasty " pasture of Kangaroo Island, begun in 1931 by Marston and his co-workers, showed that supplementing pasture in respect of calories, protein and phosphorus, did not prevent the occurrence of the disease. A transient favourable response to ground ironstone was obtained in two places, which it was thought might be due to contamination by trace elements. Feeding experiments with trace elements begun in 1934 showed that copper alone was ineffective when fed to diseased ewes, addition of 1 mg. cobalt daily to the diet of the animals receiving copper resulted in improved appetite and condition ; withdrawal of the cobalt caused the sheep to sicken again. Cobalt alone was not very effective for sick ewes. In all of the sheep in these experiments parasitism was controlled by periodic drenching with carbontetrachloride. Further experiments were carried out at Robe where the disease is more severe than on Kangaroo Island. Iron and copper administered to mature sheep affected with coast disease brought

about a temporary improvement in some but failed to prevent the disease progressing to a fatal termination. The haemoglobin in these sheep was not so low before death as in control sheep. Most of the sheep treated with copper and cobalt were restored to apparently good health. The progress of ewe weaners on "coasty" pasture was observed over a period of 54 weeks. Untreated controls developed severe symptoms and death followed in all cases. In other groups various treatments were begun after 13 weeks on the pasture. Treatment with 2 mg. copper thrice weekly had no effect, 2 mg. cobalt thrice weekly permitted growth in some animals, but in most cases failed to prevent the progressive development of anaemia. Copper and cobalt enabled satisfactory growth to be made, prevented the development of anaemia and restored mildly affected sheep to health (Marston, 1935; Lines, 1935; Marston *et al.*, 1938).

Philp *et al.* (1938–39) showed that sheep could be maintained in health for a year on "coasty" pasture on King Island by drenching with copper and cobalt or limonite. In control sheep which became "coasty" the possibility that parasitic infestation might be complicating the condition was excluded by the results of faecal egg counts. In later experiments, Philp *et al.* (1939–40) reported that treatment with cobalt alone was not very effective, but that copper and cobalt were curative as well as preventive. Scott (1938) reported that on Thistle Island, where the pasture is all "coasty" and there is no healthy "change" pasture, dosing with cobalt and copper cured coast disease and made it possible to raise lambs. Bennetts (1940) has also reported that cobalt and copper is curative and preventive of coast disease in West Australia.

Composition of the Blood. Marston *et al.* (1938) have described the progressive anaemia which is associated with emaciation in "coasty" sheep. In the final stages very low levels of haemoglobin are reached. The red cell volume is usually lower than normal, haemoglobin per cell being somewhat lower while haemoglobin per unit of cell volume remains within normal limits. Morphological changes in the red cells may be observed as the disease progresses. Some red cells appear to have a smaller diameter, others a larger diameter than normal, but changes in the red cells seem to bear little relation to the degree of anaemia. Reduction in the number of red cells appears to be the main cause of the low haemoglobin values. The presence of macrocytes, however, does serve in some measure to distinguish the anaemia from secondary anaemias due, for example, to parasitism. There was no evidence of excessive blood destruction. The progress of the anaemia in 8 sheep kept on "coasty" pasture in South Australia is shown in Table I. Bennetts (1940) records haemoglobin values ranging from 5·1 to 8·8 g. per 100 c.c. for 7 badly affected sheep in West Australia; a lamb killed in the advanced stages of the disease had a haemoglobin value of 5·7 g. per 100 c.c. and a red cell count of 5·3 million; a stray "coasty" heifer showed 6·2 g. haemoglobin and a red cell count of 3·5 million.

The effect of feeding trace elements on the haemoglobin levels of sheep on "coasty" pasture is also shown in Table 1 (Marston *et al.*, 1938).

TABLE 1

THE EFFECT OF FEEDING TRACE ELEMENTS ON THE HAEMOGLOBIN (g. per 100 c.c.) OF SHEEP ON "COASTY" PASTURE

Treatment	Co, Cu, Fe, Ni, Mn, Zn	Co only	Co + Cu	Cu only *	Control
Weeks on coasty pasture :					
2	11·1	10·7	11·5	11·2	11·0
8	10·4	9·6	10·6	9·0	10·4
*13	9·7	8·6	9·0	9·7	9·5
20	9·8	7·1	9·5	8·4	7·8
24	10·4	6·9 (7)	10·1	8·5	7·9
26	10·7	6·9 (7)	9·8	8·4	8·4
30	9·9	6·5 (7)	10·2	7·9	7·7 (7)
33	10·0	6·3 (7)	9·7	7·6 (7)	7·4 (7)
39	8·4	6·8 (7)	9·1	4·9 (4)	5·4 (6)
43	9·1	7·2 (7)	10·1	3·5 (2)	4·5 (3)
47	9·3	7·2 (7)	9·9	(nil)	1·8 (1)
54	10·2	7·4 (7)	10·3	(nil)	(nil)

* Treatment begun.

In the groups where deaths occurred the number of survivors is shown in brackets. In all other cases the values represent the mean of 8 animals. The doses of cobalt, nickel, zinc and copper were 2 mg. each 3 times weekly; of manganese, 20 mg. 3 times weekly; of iron, 40 mg. 3 times weekly.

Pooled samples of blood from each group were used for red cell counts and the results are shown in Table 2.

TABLE 2

THE EFFECT OF FEEDING TRACE ELEMENTS ON THE RED CELL COUNT (mill. per c.mm.) OF SHEEP ON "COASTY" PASTURE

Treatment	Co, Cu, Fe, Ni, Mn, Zn	Co	Co + Cu	Cu	Control
Weeks on coasty pasture :					
30	10·3	9·0	11·2	8·5	9·4
33	10·6	8·2	10·8	8·2	8·9

Bennetts (1940) took 4 lambs showing early signs of coast disease and treated 2 with cobalt and copper twice weekly from 2/12/38 to 10/8/39, leaving the others as controls. The effect on haemoglobin is shown in Table 3.

TABLE 3

THE EFFECT OF A SUPPLEMENT OF COBALT AND COPPER ON THE HAEMOGLOBIN OF "COASTY" LAMBS

		Treated Lambs		Control Lambs	
		1	2	1	2
Hb g. per 100 c.c.	20/11/38	9·3	9·6	9·3 killed 22/12/38	10·2
,,	25/1/39	14·6	17·6		5·7
,,	10/8/39	13·0	11·5		

Marston *et al.* (1938) found no abnormality in the copper content of the blood of affected sheep.

Composition of organs. Analyses by Marston *et al.* (1938) of organs of healthy and "coasty" sheep showed abnormal concentrations of iron in the liver and to a lesser extent in the spleen and kidneys of affected sheep. Histological examination indicated that this iron was largely in the form of haemosiderin. The copper content of liver, kidney and spleen was considerably reduced in coast disease, especially that of the liver, although one "coasty" sheep showed normal liver copper. Treatment with cobalt and copper reduced the iron content of the tissues to normal but did not materially increase the copper content although the sheep did well. In one sheep treated with iron and copper the iron content of the organs was reduced. Bennetts (1940) also reported haemosiderosis of the spleen, and to a lesser extent of the liver and kidneys of affected sheep in West Australia.

All the workers on coast disease conclude that the condition is a dual deficiency of cobalt and copper. The existence of a copper deficiency in these areas seems to be further substantiated by the simultaneous occurrence of ataxia in lambs in certain "coasty" areas of South and West Australia (Marston *et al.*, 1938; Bennetts, 1940) and straight steely wool in sheep in certain parts of South Australia (*J. Dept. Agric. S. Austral.*, 1941) since both these conditions are said to be due to copper deficiency (see pp. 38–42 and 50).

COMPOSITION OF THE SOIL AND PASTURE

Soil. Philp (1933) found that the coastal dunes of Tasmania were very high in calcium and were also high in iron and phosphorus. He suggested that the excess of calcium might inhibit the assimilation of iron and phosphorus. Thomas (Marston *et al.*, 1938) analysed samples of affected South Australian calcareous soil for major constituents and stated that the idea of cobalt deficiency in soil and pasture was not inconsistent with the geochemistry of the affected areas. Philp (1939–40) states that the soil of experimental plots in "coasty" areas of King Island showed a deficiency of cobalt. The results given by Dumaresq (1943) were as follows: the cobalt, nickel and copper contents of soils from eight localities in King Island ranged from 0·0 to 3·9 p.p.m. of cobalt, 0·0 to 39·2 p.p.m. of nickel and 2·6 to 17·1 p.p.m. of copper. The values for the calcareous sand soil on which "coasty" experiments were made were 0·0, 0·6 and 17·1 p.p.m. respectively. Analyses of samples of soil from a "coasty" paddock after topdressing with 20 lb. copper sulphate and 2 lb. crude cobalt sulphate per acre gave values ranging from 0·72 to 1·5 p.p.m. of cobalt in dry soil, with the exception of 2 values of 17·5 and 38·0 p.p.m. from samples which were considered to have become contaminated in some way.

Pasture. Philp (1939–40) found that sheep could be maintained in perfect health in "coasty" areas on King Island by topdressing the pasture with copper and cobalt sulphates. The results of analyses of pasture for cobalt given by Dumaresq (1943) were as follows: a sample of pasture from the untreated area showed a cobalt content of 0·10 p.p.m. D.M. and a sample taken from the treated area 3 months after the application of cobalt and copper showed a value of 0·78 p.p.m. D.M. It is stated that both samples were slightly contaminated by soil, correction for which would not appreciably affect the value of the treated pasture but would probably reduce the value of the untreated sample to a level which is often associated in New Zealand with stock ailment of the cobalt deficiency type. Both samples were collected at the end of September 1939. According to Bennetts *et al.* (1942) Dumaresq has stated in a private communication that the "coasty" pastures of King Island where "falling disease" also occurs are low in copper (see p. 49).

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"SALT SICK." FLORIDA

Occurrence and Description of the Disease

A wasting disease, known locally as "salt sick" and by other colloquial names, occurring among cattle on certain range areas of the gulf coast and coastal plains was mentioned in the Florida Agricultural Experiment Station Bulletin as early as 1888. Becker *et al.* (1931) and Neal and Becker (1933) described the soils of the affected areas as consisting of white and grey sands, cumulose muck lands and shallow residual soils overlying marl. Healthy ranges were on red and yellow sands, clays, shallow sands overlying clay and soils subject to overflow from clay lands. Later Bryan and Becker (1935), investigating the soils in the "salt sick" areas, found them to consist of fine sands of the Leon, Portsmouth, Dade and Norfolk series and also peat deposits. Adjacent healthy areas consisted of fine sandy loams or sands with clay subsoil. The average content of silt and clay in the surface soil of 21 mineral soils where "salt sick" occurs was only half that of 17 mineral soils in healthy areas.

The disease occurs mainly in cattle, but sheep, goats and swine are reported to be similarly affected. "Salt sick" cattle exhibit almost complete loss of appetite, emaciation, weakness and pallor of the mucous membranes. Depraved appetite is sometimes observed. Growth is retarded and sexual maturity delayed. Parasites may aggravate the condition but are not necessarily associated with it. The blood is said to be pale in colour and the total volume is greatly reduced. Affected animals have generally, but not always, a lower concentration of haemoglobin in the blood than healthy animals. The main finding at *post-mortem* examination is lack of body fat; atrophy of the spleen is also observed.

The condition occurs more frequently among cattle over 6 months of age and in heifers just after the first calving. It has been noted in cattle of all ages kept for a long time on affected ranges, but mortality is greatest among young cattle. There is little evidence of seasonal difference in incidence.

A practical means of correcting the "salt sick" condition is to remove cattle from affected areas, after 6 to 10 months grazing, to healthy areas where they recover. They may then be returned to graze on the affected area for another 6 to 10 months' period (Becker *et al.*, 1931).

Experimental Studies

Feeding experiments. Neal *et al.* (1931) and Becker *et al.* (1931) found that when affected cattle were given iron and copper treatment the condition was overcome in all but advanced cases, but iron supplements alone proved inadequate. Bonemeal did not effect recovery (Becker *et al.*, 1931–32).

Neal and Ahmann (1937 [1]) pointed out that although treatment with iron and copper was effective in some areas, in others cobalt was also necessary for normal growth and function. They found that calves pen-fed on a basal ration consisting mainly of foods grown on " salt sick " land lost appetite, became emaciated and listless and showed poor coats and retarded sexual development. Calves fed a supplement of iron oxide plus copper developed these symptoms sooner than those on the basal ration, but calves fed a supplement of cobalt, or cobalt, iron and copper, did well. The most striking effect of the cobalt was the return of appetite (Neal and Ahmann, 1937 [2]). Becker *et al.* (1931–32) point out that although parasites may aggravate the condition " salt sick " has been produced in parasite-free calves under controlled feeding conditions.

Composition of the blood. Neal and Becker (1933) investigated the haemoglobin in the blood of healthy and " salt sick " cattle in the same herds (Table 1).

TABLE 1

Haemoglobin in the blood of Healthy and " Salt Sick " Cattle

Group	No. of Animals	Type	Hb g. per 100 c.c. (Daré)	
			Range	Mean
Healthy cattle	2	Cattle	9·62–13·74	11·68
	28	Yearlings	10·72–16·49	13·34
	4	> 2 years	11·68–14·43	13·57
" Salt sick " cattle from same herds	12	Calves	3·02– 9·62	5·92
	96	Yearlings	3·44–13·05	8·38
	34	> 2 years	4·81–13·19	9·04
	2	> 12 years		4·81
" Salt sick " cattle shortly before death	9	Calves	1·37– 9·62	4·49
	7	Yearlings	3·44–12·37	6·62

Although, in general, the affected animals had a lower concentration of haemoglobin in their blood than had healthy animals, a number of affected cattle had quite high haemoglobin values. Changes in the blood volume and other physiological adaptations affect the concentration of haemoglobin in the blood to such an extent that, even after allowing for normal variations, haemoglobin concentration alone cannot be regarded as an exact measure of changes in total haemoglobin.

Becker *et al.* (1931–32) found that the average haemoglobin of 87 visibly affected cattle was 8·28 g. per 100 c.c., and after 1 month's treatment with iron and copper, which improved their condition, the average haemoglobin was 10·61 g. per 100 c.c. blood ; healthy cattle in the same herd averaged 13·86 g. haemoglobin. Neal and Becker (1933) reported that cattle with haemoglobin as low as 3·02 g. per 100 c.c. recovered following treatment with iron and copper. Neal and Ahmann (1937 [2]) found no correlation between the haemoglobin and the condition of calves in their experiment with calves pen-fed on fodder from " salt sick land." Often there was apparently an increase in haemoglobin level when a calf lost condition. There was no level so low as to be classed as anaemic. In general, however, the concentration of haemoglobin per cell and per cell volume was higher in animals receiving cobalt as a supplement to " salt sick " fodder. The individual cell was larger in cobalt fed calves. Animals not receiving cobalt showed morphological changes in the red cells and an increased proportion of leucocytes to erythrocytes.

Composition of organs and other tissues. Haemosiderosis of liver and spleen was observed in affected calves fed rations from " salt sick " land (Becker *et al.*, 1931). Rusoff (1941) analysed tissues and organs of newborn calves from " salt sick " and healthy dams. With the exception of the skeleton nearly all the tissues of a calf from a " salt sick " dam contained more copper than comparable tissues from a calf with a normal mother. It is stated that copper storage by the calf was not influenced by " salt sickness " in the dam nor was the calf " salt sick." It is suggested that some other factor may be required for copper utilisation. Analyses of organs for other trace elements including cobalt showed no significant difference between the calves of " salt sick " and healthy mothers.

Composition of the Soil and Pasture

Soil. Bryan and Becker (1935) investigated 21 mineral soils on which range cattle developed " salt sick," 2 " salt sick " areas on peat and 17 mineral soils in healthy areas. The healthy sandy soil ranges contained on the average in the surface soil 10 times as much iron, twice as much copper,

5 times as much phosphorus and 5 times as much calcium as the "salt sick" areas. The organic soils, although "salt sick," had high calcium, iron and copper contents. The values for copper content were as follows :

"Salt sick" mineral soils . .	1·50 to 7·85 p.p.m.	average 3·97 p.p.m.
Healthy mineral soils . .	2·42 to 20·97 ,,	,, 8·56 ,,
"Salt sick" peat soils . .	12·75 and 42·30 ,,	

Low values for copper in certain soils in Florida have been reported by Rogers *et al.* (1939) and Peech (1939) (quoted by Maynard, 1941).

Pasture. Analyses of wire grass, *Aristida purpurascens*, the predominant plant on Florida ranges, showed that the grass on "salt sick" areas tended to be lower in calcium, phosphorus, magnesium and iron than grass on healthy areas. Other forages and grains analysed also showed the same tendency (Becker *et al.*, 1931–32 ; Neal and Becker, 1933 [2]). Neal and Ahmann (1937 [2]) found that a supplement of cobalt was needed to keep calves healthy on a ration of crops grown on "salt sick" land. They were unable to detect cobalt spectrographically in the ash of any of the feeds used, but believed that since Rusoff *et al.* (1937) were unable to detect cobalt spectrographically in wire grasses from "salt sick" or healthy areas, the usefulness of the method for detection of small amounts of cobalt was open to doubt. Rusoff *et al.* (1937) also analysed the wire grasses spectrographically for copper. The values ranged from 4·7 to 10·1 p.p.m. D.M. and those from "salt sick" areas did not differ from those from healthy areas.

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CHAPTER III

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO DEFICIENCY OF COPPER

INTRODUCTION AND DISCUSSION

The first published report suggesting that deficiency of copper occurs naturally in livestock appears to be that of Neal and associates in 1931. These workers observed that cattle suffering from a wasting disease, often accompanied by anaemia, known as "salt sick" in Florida, responded to treatment with iron oxide and copper but not to iron oxide alone. Subsequent investigation showed that the disease was primarily a cobalt deficiency associated with deficiency of copper. The iron oxide which, with copper, was curative, probably contained traces of cobalt.

In 1933 Sjollema in Holland reported that liksucht of cattle, a wasting disease, sometimes accompanied by anaemia, and characterised by depraved appetite, could be cured by administration of copper sulphate. Shortly afterwards Marston and his associates began experimenting with trace elements in the treatment of sheep suffering from coast disease, a wasting disease with anaemia in the later stages, which affects cattle and sheep in parts of Australia. They found that sick sheep responded to cobalt feeding but, as in the case of "salt sick" cattle in Florida, copper also was essential for complete recovery. In these "coasty" areas lambs developed a form of ataxia, and administration of a mixture of trace elements to the gestating ewe prevented the occurrence of coast disease in the ewe and ataxia in the lambs. These results were not published until 1938. Meanwhile, Bennetts and Chapman had been for some years investigating ataxia in unweaned lambs in parts of Western Australia where coast disease did not occur. They found that the disease could be prevented by feeding copper sulphate to the gestating ewes and the progress of the disease could be arrested by giving copper sulphate to lambs already affected. Marston and his associates subsequently applied these findings with equal success to the control of ataxia in the coast disease areas of S. Australia.

Bennetts and his co-workers also investigated the condition of adult sheep in flocks affected with ataxia, but free from typical symptoms of coast disease. They found a characteristic type of "stringy" wool in sheep of any age and either sex. Breeding ewes frequently showed anaemia which disappeared after lactation and showed severe persistent diarrhoea and loss of condition during the wet months. All responded to supplementary copper feeding. There appeared to be a positive correlation between severe anaemia in the ewe and the incidence of acute ataxia, although subacute

cases of ataxia occurred in lambs whose mothers showed no evidence of anaemia. Bennetts and his co-workers emphasise that there is no sign of cobalt deficiency in this area and no lack of cobalt in the livers of sheep, and that the anaemia in these ewes differs in some respects from that observed in coast disease, and is entirely different from that described by Filmer in uncomplicated cobalt deficiency.

"Swayback," a disease similar to the enzootic ataxia of Australia, is widespread in Britain. Investigations in Derbyshire by Dunlop and Wells have shown that "swayback" can be controlled by giving copper to the gestating ewes. Adult sheep in these areas do not show signs of copper deficiency. Anaemia occurs only occasionally in the ewes, and is not related to the occurrence of "swayback" in the lambs. Shearer and his co-workers have suggested that the condition in the lambs may be due to impaired transference of copper from the ewe to the foetus. Recent investigations in Peru show that the disease known as "renguerra" is identical with "swayback," and is controlled by administration of copper to the gestating ewe.

In Holland, Sjollema and Brouwer have described a disease in cattle characterised by severe and persistent scouring which responds to treatment with copper. Brouwer points out that although diarrhoea frequently occurs towards the end of "liksucht" it is not there a dominant symptom throughout as in the scouring disease. According to Brouwer, haemoglobin levels are usually low in "scouring" cattle, but Sjollema found no anaemia in the cases he investigated.

In parts of Australia where ataxia in lambs (but not coast disease) is enzootic, foals and calves show signs of malnutrition and structural and postural defects, and cattle lose condition, become anaemic and frequently show depraved appetite. According to Bennetts, the clinical picture in cattle is similar to that of the scouring disease described by Brouwer and Sjollema, except that in Australia, although diarrhoea occurs, persistent scouring is not characteristic of the disease. An improvement in the health of cattle, calves and foals follows treatment with copper. An unusual feature of this area is that cattle suddenly drop dead while still apparently in moderately good condition. Bennetts claims to be able to prevent this "falling disease" or "sudden death" by administration of copper, and considers it to be the terminal phase of acute copper deficiency.

A condition resembling the scouring disease of Holland occurs in cattle and, to a less extent, in sheep on the "teart" pastures of Somerset, and Ferguson *et al.* have found copper sulphate curative and preventive. In both of these scouring diseases depigmentation of the hair of brown and black cattle is observed. Since the hair of rats on an experimental diet deficient in copper becomes depigmented (McCollum *et al.*, 1939) this might be taken as presumptive evidence of copper deficiency in the cattle and, in fact, copper restores coat colour as well as curing the scouring in both areas.

In most of these diseases which respond to treatment with copper, the diagnosis of copper deficiency has been confirmed by analyses of tissues, blood and milk. Low reserves of copper have been found in the livers of lambs and ewes in areas where ataxia and "swayback" are enzootic, in the livers of cattle in "falling disease" areas and those suffering from scouring disease in Holland, and in the livers of "salt sick" cattle and coasty sheep. Low levels of blood copper are frequently found, especially in the later stages of the diseases, but animals appear to maintain their blood copper levels as far as possible by drawing on the reserve of copper in the liver. The copper content of the milk of the mothers of ataxic lambs and of cows in "falling disease" areas is lower than normal, and the hair of cattle suffering from scouring disease in Holland is low in copper.

No similar study has been made in the "liksucht" disease of Holland, or in the scouring disease of Somerset.

These findings imply lack of copper in the pasture grazed by the affected animals, but only in some areas has this been found.

Sjollema confirmed his diagnosis of copper deficiency in "liksucht" by demonstrating that hay from the affected areas was low in copper, usually containing less than 3 p.p.m. D.M. Similar low values have been reported for Australian pastures upon which ataxia in lambs, "stringy" wool in sheep and "falling disease" in cattle are enzootic. In general Beck found that these conditions were prevalent on pastures containing less than 3 p.p.m. D.M. of copper; values between 3 and 5 p.p.m. were marginal, and values over 5 p.p.m. indicated sound land. Seasonal variations in the incidence of ataxia were not always correlated with seasonal differences in the copper content of the pasture, and Beck suggests that the availability of the plant copper to the animals may depend on seasonal factors as yet not fully understood. For instance, in areas where ataxia is enzootic a season favouring a rank growth of capeweed seems to favour the occurrence of ataxia, not because capeweed has a low content of copper compared with other pasture species, but it has been suggested, because the scouring in breeding ewes which occurs in such seasons reduces their absorption of copper.

A survey of the copper content of Swedish pastures shows that at Gisselas the copper content of 40 samples of hay averaged only 2 p.p.m. D.M., but the occurrence of licking disease is rare because of the practice of feeding molasses containing 14–16 p.p.m. of copper.

In Holland where "liksucht" occurs in cattle, cereals also suffer from a disease known as "re-

clamation disease," which can be cured by fertilising with copper sulphate. This plant disease occurs also in the copper deficient areas of Australia. Topdressing with copper sulphate, which cures " reclamation disease," also raises the copper content of pastures and controls the incidence of ataxia.

In contrast to the Australian pastures, " swayback " and " teart " pastures in Britain are well supplied with copper, and the copper content is not related to the incidence of these diseases. In the " swayback " investigations the copper content of samples of " affected " and unaffected pasture ranged from 12 to 27 p.p.m., and Ferguson and his co-workers found values of 11 to 18 p.p.m. D.M. of copper in " teart " and non-" teart " herbage.

In Holland Brouwer found no relation between the incidence of scouring disease and the copper content of the pastures, which, although not as well supplied with copper as " teart " pastures, usually contained more than 5 p.p.m. D.M. and would be considered sound by Australian standards. Similar values have been reported for the copper content of samples of the principal grass in Florida pastures, taken from both " salt sick " and healthy areas. In these two areas the number of samples analysed is small, and it is possible that more extensive sampling might reveal a lack of copper in the pasture. This could hardly be expected in the " swayback " and " teart " areas where all the values obtained were high.

If the low copper reserves and the beneficial effect of copper supplements in " swayback " are to be taken as evidence of copper deficiency, then there remains the possibility that some unknown factor affects the utilisation of dietary copper. Shearer *et al.* have suggested that the high calcium to phosphorus ratio or the high lead or zinc content of these pastures may affect the absorption of copper. Tompsett (1940) has shown that the absorption of copper by mice on a high calcium diet is lower than on a low calcium diet, but whether this would be true for sheep in the circumstances obtaining in " swayback " areas is not known.

In contrast to the lack of precise knowledge of the cause of " swayback," the cause of " teartness " has been established by Ferguson and his associates as due to an excess of molybdenum in the pasture. This has been confirmed by the artificial production of scouring disease on healthy pasture by feeding sodium molybdate or by raising the molybdenum content of the pasture by topdressing with sodium molybdate. No explanation of why copper sulphate should act as an antidote in molybdenum poisoning has yet been suggested, but it may be that an abnormal intake of molybdenum interferes with the utilisation of copper. This might explain the similarity of the syndrome, including the depigmentation of hair, to that described by Brouwer in Holland where there is no excess of molybdenum in the pasture. Analyses of liver for copper in the teart areas are needed to confirm or dispose of the view that this trouble is a conditioned copper deficiency.

Finally, there is yet another disease, the " lecksucht " of Schleswig-Holstein, attributed by some investigators to deficiency of copper. The evidence here is conflicting. Its association with " reclamation disease " in plants and the beneficial effect of topdressing with copper sulphate on both plant and animal diseases, together with the low values for copper in some, but not all, samples of " affected " pasture, indicate the possibility of copper deficiency. The work of Krzywanek and Brüggemann shows that some, but not all, " lecksucht " hays are well supplied with copper. A healthy ox fed a " lecksucht " hay containing only 2·5 p.p.m. D.M. of copper showed a negative copper balance until the hay was replaced by another " lecksucht " hay containing 4 p.p.m. A copper content of 2·5 p.p.m. D.M. is definitely deficient by Australian standards. On the other hand, no clear relationship has been established between the incidence of the disease and the copper content of the pastures, nor has the disease been successfully controlled by direct administration of copper. Further information is needed on the effect of feeding larger doses of copper than have yet been tried, and on the copper content of the organs and pasture in this area.

From this summary it appears that copper deficiency occurs in cattle and sheep, and to some extent in foals, as the result of lack of copper in the pastures. It seems also that a conditioned copper deficiency may occur on pastures well supplied with copper because of factors affecting the availability of the copper to the animals.

The occurrence of anaemia and excessive deposition of iron in the liver and spleen in cattle and sheep suffering from uncomplicated copper deficiency due to lack of copper in Australian pastures indicate that in ruminants as in rats, pigs and probably other non-ruminants, copper is essential for the utilisation of iron for haemoglobin formation. Histological studies in Australia have shown large deposits of iron in the form of haemosiderin, a degradation product of haemoglobin, and since Bennetts and Chapman found no evidence of excessive blood destruction, copper is probably essential for the re-utilisation of iron released by the breakdown of haemoglobin.

Deposition of haemosiderin has also been reported to be excessive in " salt sick " cattle, in scouring cattle in Holland, and in sheep suffering from coast disease where it was reduced to normal by treatment which copper and cobalt, and in one instance by iron and copper.

In enzootic marasmus, in which affected animals show low reserves of copper, although the disease

itself is cured by cobalt alone, deposition of haemosiderin is said to have been reduced to normal by cobalt administration. The absence of reports of haemosiderosis in cobalt deficient animals in New Zealand, where the intake of copper is adequate, does not support the view that cobalt is essential for the re-utilisation of iron released by breakdown of haemoglobin. Further investigation of this point is required in view of the work of Schultze *et al.* (1936), which shows that the abnormally high reserves of organic iron in the liver and spleen of pigs fed iron after being made anaemic through lack of iron and copper, is reduced to normal by administration of copper.

The occurrence of other symptoms before the appearance of anaemia in copper deficiency, and in some cases the non-appearance of anaemia, suggest that the function of copper in ruminant nutrition is not restricted to its haematopoietic effect. The variation in symptoms from one species to another, and in the same species from one age to another, and from one area to another, may be due to differences in the function of copper in different species and to differences in degree of deficiency between one area and another, but it seems probable that other factors also contribute to the clinical pictures observed. For instance, in "swayback" the high lead content of the pastures is reflected in the high storage of lead in the tissues of ewes and lambs, and although there is no evidence of plumbism in the ewes, it is possible that lead is mobilised from the skeleton during pregnancy as in women (Hansmann and Perry, 1940), passes through the placenta, and contributes to the clinical picture in the lamb which, according to Shearer and his co-workers, is not unlike that of young animals suffering from lead poisoning.

References, other than those listed below, will be found in the more detailed report of the investigations in each area which follows.

[Since this review was published a report of copper deficiency in sheep and cattle in New Zealand has appeared (Cunningham, 1944). We take the opportunity provided by the reprinting of the T.C. to insert a brief reference to this interesting work.

The condition in cattle is known as "peat scours" because of the type of soil upon which it mostly occurs and because severe scouring is a characteristic symptom. An interesting feature is the depigmentation which occurs in the hair of black cattle kept for long periods on "affected" land (*cf.* scouring disease in Holland and on the "teart" pastures of Somerset). In the areas where cattle are affected lambs suffer from enzootic ataxia. Investigations have shown that the levels of copper in the blood and livers of affected animals are low. The pasture is poorly supplied with copper, levels as low as 2 to 5 p.p.m. of copper on a dry matter basis being recorded. The condition has been controlled by direct administration of copper to the livestock or by topdressing the pastures with fertilisers containing copper.]

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ENZOOTIC ATAXIA. AUSTRALIA

Occurrence and Description of the Disease

The name enzootic ataxia was first applied by Bennetts (1932) to a disease in lambs known popularly as "Gingin rickets," after the district in which it was first recognised and where in the first decade of the twentieth century it began to constitute a real problem. The distribution of the disease in West Australia has recently been summarised by Bennetts and Beck (1942). The affected areas are Gingin district, an area of approximately 30,000 acres; Dandarragan, a district of approximately 200,000 acres; properties near Mt. Barker; semi-coastal country bounded by Ludlow and Forrest Grove and extending inland for 10 to 20 miles where "falling disease" in cattle is also a problem; and coastal country as far north as Broome, various points along the west coast to the south, and the southern coast as far west as Doubtful Bay, where ataxia is associated with "coast disease." The occurrence of ataxia in lambs in the State of South Australia, Victoria and Kangaroo Island, sometimes associated with "coast disease," has been reported by Marston *et al.* (1938).

Enzootic ataxia occurs on a variety of soils. The Gingin and Dandarragan soils are derived from the same Cretaceous series, and according to Bennetts (1935) the main soil divisions in the Gingin area are renzina, ferruginous sandstone and green sands. The soil of the affected coastal areas in West Australia is derived largely from wind-borne shell fragments and consists of sand rich in lime or of consolidated limestone. Beck (1941 [3]) mentions that ataxia has been confirmed in Dongarra in the zone of red brown earths, and describes the soils of Pardelup Prison Farm at Mt. Barker, where ataxia occurs, as consisting largely of immature granite and lateritic gravelly types. In South Australia ataxia in lambs occurs most commonly on calcareous soils of Recent origin (Marston *et al.*, 1938). It also occurs on the limestone area of south-eastern South Australia.

The disease occurs in the unweaned lambs of ewes grazing on the " affected " pasture. It most commonly affects lambs from 1 to 2 months old. They become unthrifty and stiff in gait and growth is retarded. When driven, the lambs develop ataxia. There is no anorexia. The disease progresses fairly rapidly and lambs usually die within 3 or 4 weeks. In those that do not develop the disease until they are 3 or 4 months old the course is generally less severe ; such lambs almost always survive. An acute type of the disease sometimes affects lambs from birth to 4 or 5 weeks old. The development of muscular inco-ordination is rapid and the lambs usually die within 3 or 4 days ; those less severely affected may live longer and show the typical ataxic gait.

Ataxia has been observed in several breeds, and there is little direct evidence to support the view of some stock owners that the progeny of the Merino ewe is more susceptible than that of the cross-bred. According to Bennetts and Beck (1942) there is no definite seasonal varation in the incidence of the disease, but lambs dropped in the late spring or dry summer months are less liable to develop the disease than those born at the usual time, between May and July, although the mothers may have been on the " affected " country for the same length of time. It is pointed out that this may be related to the slower growth rate of summer lambs. Characteristic lesions of the spinal cord in ataxic lambs have been observed by Bennetts (1932, 1933) in West Australia and by Marston *et al.* (1938) in South Australia. Gross brain lesions of the type described by Innes in " swayback " of lambs in Derbyshire (see p. 43) were not observed by Marston *et al.* (1938) and were seen only in very acute cases of ataxia by Bennetts and Beck (1942). Marston *et al.* (1938) reported that haemosiderosis of the liver and pancreas was a constant finding in cases of long standing examined in Victoria and South Australia, and Bennetts and Beck (1942) reported haemosiderosis of the liver in 8 out of 9 affected lambs over the age of 5 weeks.

Anaemia, although commonly present in subacute cases, is not a constant feature of the disease. Since enzootic ataxia affects only unweaned lambs and can be controlled by weaning on to pasture it is obvious that the condition of the ewes is of crucial importance.

In flocks affected with ataxia adult sheep show stringiness of wool. Anaemia, diarrhoea and loss of condition are signs exhibited by breeding ewes, and haemosiderosis of the liver, kidney and spleen has been demonstrated. According to Bennetts and Beck (1942) the anaemia is exhibited only by the breeding ewes and recovery is generally rapid following the cessation of lactation. Severe anaemia in the ewe is correlated with the more acute form of ataxia in the lambs, mothers of subacute cases may show no evidence of anaemia.

The methods of management which control the incidence of ataxia in lambs have been summarised by Bennetts and Beck (1942). The occurrence of ataxia may be prevented by using only breeding ewes introduced from healthy districts immediately prior to mating. If breeding ewes are introduced much earlier affected lambs may result. A breeding flock may be maintained on affected properties provided ewes are transferred during their gestation to known healthy country. The minimum effective period varies according to locality from 4 to 8 weeks. Alternatively, a change to healthy country after parturition may be found effective unless the course of the disease is very acute. According to Bennetts (1935 [2]) lambs can be reared successfully on the milk of cows grazing on the affected area if taken away from their mothers in the early stages. Lambs which show the first symptoms only after reaching the age of about 3 months may be reared successfully if weaned on to the pasture grazed by the ewe.

Experimental Studies

Feeding experiments. Since the disease occurred in sucking lambs only, it was attributed at first to the presence of some toxic element in the milk. Chemical analyses indicated a somewhat higher content of lead in the milk of ewes with affected lambs than in that of normal ewes, and this evidence seemed to support the view that the symptoms of the disease in lambs resembled in some ways those of lead poisoning. Ammonium chloride was administered to ewes as a deleading agent with results beneficial to the lambs (Bennetts, 1935,[1] 1935 [2]). Later, Bennetts and Chapman (1937) found that if the ammonium chloride used was a pure analytical reagent it had no beneficial effect. The milk of ewes rearing healthy lambs was found to be higher in copper content than that of mothers of ataxic lambs, and in preliminary experiments Bennetts and Chapman found that administration of copper to gestating ewes prevented anaemia and the occurrence of ataxia in the progeny, and that copper given to ataxic lambs arrested the progress of the disease. This was further confirmed by Bennetts and Beck (1942), and Marston *et al.* (1938) reported similar success with copper treatment in South Australia.

Composition of the blood. Anaemia is not a constant feature of enzootic ataxia. Bennetts and Beck (1942) examined the blood of 4 acute cases of ataxia in lambs aged less than 8 days and found no evidence of anaemia. The blood pictures of normal and affected lambs, as described by them, are compared in Table 1.

TABLE 1

Blood Picture of Ataxic and Normal Lambs in West Australia

Subjects	Age: Weeks	Hb g. per 100 c.c.	R.B.C. mill. per c.mm.	Cell Vol. per cent.	Mean Red Cell Vol. c.μ.	Colour: Count Ratio	Colour: Volume Ratio
12 healthy lambs from sound areas . .	8–18	13·2–17·6 (15·7)	14·0–16·9 (15·0)	41·3–47·5 (44·5)	27·0–32·1 (29·6)	0·88–1·2 (1·05)	0·29–0·43 (0·35)
3 ataxic lambs . .	6–12	5·9–13·2 (8·5)	9·4–17·1 (12·1)	18·8–37·5 (25·4)	20·0–21·9 (20·8)	0·60–0·77 (0·68)	0·30–0·35 (0·33)

Stippled cells were rarely observed and were never numerous.

Filmer is reported by Bennetts and Beck (1942) as having found the mean red cell diameter of 9 ataxic lambs to be somewhat less than that of 7 normal lambs. Bennetts and Beck (1942) conclude that the limited data available indicate that the anaemia which occurs is of the microcytic hypochromic type. The blood pictures of 17 lambs with ataxia in South Australia have been recorded by McDonald (1942) who agreed that, although anaemia may be present, it is not an essential part of the syndrome. (Table 2.)

TABLE 2

Blood Picture of Ataxic Lambs in South Australia

Hb g. per 100 c.c.	R.B.C. mill. per c.mm.	Cell Volume per cent.	Mean Cell Volume c.μ.	Colour: Count Ratio	Colour: Volume Ratio
9·4–12·7 (11·5)	9·8–19·0 (13·8)	31·3–48·8 (37·8)	23–33 (28)	0·63–1·06 (0·85)	0·26–0·33 (0·30)

Anaemia frequently occurs in gestating ewes in areas affected with ataxia (Bennetts and Beck, 1942). Haemoglobin may be considerably decreased, commonly to below 6 g. per 100 c.c., and values as low as 3·7 g. have been observed. There is a corresponding gross reduction in red cell count, which is frequently below 6·0 million and sometimes as low as 2·7 million per c.mm. At lower cell counts morphological abnormalities of the red cells are observed. In severe cases macrocytes may be sufficiently numerous to raise mean cell volume above normal. Colour : volume ratios indicate that the saturation of erythrocytes with haemoglobin is in general somewhat incomplete (Table 3).

TABLE 3

Blood Picture of Anaemic and Healthy Sheep

Sheep Condition	No.	Hb g. per 100 c.c.	R.B.C. mill. per c.mm.	Cell Volume per cent.	Mean Cell Volume c.μ.	Colour: Count Ratio	Colour: Volume Ratio
From healthy areas	10	9·8–16·8 (12·8)	7·7–13·2 (10·4)	30·0–48·8 (36·5)	30·4–39·0 (35·2)	1·1–1·4 (1·2)	0·33–0·43 (0·35)
Anaemic from ataxic areas . .	13	3·9–7·5 (5·9)	2·7–7·4 (5·05)	15·5–24·9 (20·9)	30·4–58·5 (43·3)	0·9–1·6 (1·2)	0·22–0·32 (0·27)

In a study of 6 ewes Bennetts and Beck (1942) found that haemosiderosis was most abundant, particularly in the liver and kidneys, in subjects that had been affected with anaemia for some months.

An extensive study of blood copper in sheep during 1938–39 when the incidence of ataxia, unfortunately for the experiment, was unusually low, is reported by Bennetts and Beck (1942). No correlation between anaemia in the ewes and the subacute form of ataxia in the lambs was observed, and no definite correlation was revealed between haemoglobin and blood copper in the ewes. In general, anaemia occurred in ewes when the blood copper fell to 0·01 mg. per cent. or lower, and such low values in ewes near parturition usually resulted in ataxia in the lambs. Beck (1941 [2]) has shown that a rise in blood copper generally takes place in late spring in sheep in these areas. Values below 0·05 mg. per cent. were rarely found in ewes receiving copper supplements. There was some evidence of positive correlation between the blood copper values of lambs and their mothers. Values for 4 ataxic lambs ranged from 0·01 to 0·02 mg. per cent. Lambs on " affected " pasture topdressed with copper had blood copper values of 0·07 to 0·11 mg. per cent. ; similar values were observed in lambs

receiving a copper lick, while in control lambs blood copper ranged from 0·004 to 0·07 mg. per cent. Earlier, Marston *et al.* (1938) had found the copper levels of blood in ataxic lambs in South Australia within normal limits.

Composition of organs. Bennetts and Beck (1942) reported that liver copper values for lambs and ewes from affected properties was constantly below 15 p.p.m., and that corresponding values for healthy lambs were 120 to 350 p.p.m. and for healthy adult sheep 113 to 800 p.p.m. Although the ewes with the lowest liver copper values produced ataxic lambs, some ewes with low liver copper values produced healthy lambs. A four-week old lamb on " affected " pasture topdressed with copper had a liver copper level of 164 p.p.m. In general, there was a positive correlation between liver and blood copper in the ewes. Marston *et al.* (1938) found values of 2·6 to 9·8 p.p.m. for the copper content of the livers of ataxic lambs in South Australia.

Composition of ewe's milk. It was noted above that the milk of mothers of affected lambs, which had at first been suspected of containing a toxic amount of lead, was later shown by Bennetts and Chapman (1937) to contain less copper than the milk from the dams of healthy lambs. The copper content of ewe's milk, sampled within 3 weeks of lambing, ranged from 0·03 to 0·05 mg. per litre where the lambs were ataxic compared with values of 0·12 and 0·19 mg. for 2 ewes only recently depastured on the affected area. Beck (1941 [1]) showed that in healthy ewes the copper content of the milk fell from values of 0·20 to 0·64 mg. per litre in early lactation to 0·04 to 0·16 mg. several months later. Milk from ewes grazing on " affected " Gingin pasture had lower concentrations of copper several months after lambing, of the order of 0·01 to 0·02 mg. per litre. Higher values, 0·09 to 0·11 mg., were given by ewes grazing on this " deficient " pasture topdressed with copper.

Bennetts and Beck (1942) emphasise that there is no clinical evidence of cobalt deficiency at Gingin where they investigated ataxia. The cobalt content of 5 sheep livers from Gingin was 0·06 to 0·17 with a mean of 0·12 (on dry basis) and further, the anaemia described by Marston *et al.* (1938) in " coasty " sheep and by Filmer (1933) in enzootic marasmus are not the same as that observed in the mothers of ataxic lambs at Gingin.

Composition of the Soil and Pasture

Soil. No analysis of soil in relation to the incidence of ataxia seems to have been made, but Teakle *et al.* (1940), investigating at Dandarragan crop failures which could be prevented by topdressing with copper sulphate, found the surface soil to contain 1 to 2 p.p.m. copper (dry basis). The phosphate status of the soils is said to be considerably better than that of most West Australian soils, and in this respect these soils resemble some of the better soil types at Gingin.

Pasture. Bennetts (1935) states that the outstanding characteristic shown by analyses of Gingin pasture made by the Government Analyst, 1930–31, was the unusually high calcium and phosphorus contents. Samples obtained from plots representing the main soil types gave values for calcium oxide of 1·53 to 2·91 per cent. D.M., with the exception of one " unbelievably " high value of 5·32 per cent. He suggested, in view of the apparent resemblance of the disease to lead poisoning, that the high calcium content of the pasture ingested by the ewes caused an associated storage of lead which, while not affecting the ewes, passed out in the milk to poison the sucking lambs. Later, Bennetts and Chapman (1937), in view of clinical and pathological evidence, suggested a copper deficiency in pasture and soil, but analyses of 2 samples of Gingin pasture in October showed the high copper contents of 23 and 35 p.p.m. Later investigations, however (Beck, 1941 [3]; Bennetts and Beck, 1942), showed that mixed pasture from the ataxic areas in Gingin were low in copper, 1·1 to 3·9 p.p.m. D.M., with the exception of one value of 6·1 p.p.m., while most samples of normal West Australian mixed pasture contained 7 to 12 p.p.m. of copper.

The copper requirements of different plant species appear to be different. Elliott (1939) has pointed out that the " stalling " of subterranean clover and replacement by drooping flowered clover, which is characteristic of the affected semi-coastal area, is associated with the different copper requirements of the two species (Bennetts and Beck, 1942). Samples of clover taken from an area in the Margaret River district where this replacement was occurring gave the low values of 1·1 and 1·2 p.p.m. D.M. for copper content, compared with a value of 2·9 p.p.m. D.M. for a composite sample from the same paddock (Beck, 1941 [3]).

Analyses of capeweed by Beck (1941 [3]) indicate that, under certain conditions which are not understood, this species is able to absorb more copper from the soil than most others. It should not be used, therefore, as an indication of the availability of copper for other plants. The copper content of capeweed in ataxic districts of West Australia ranged from 3·0 to 7·7 p.p.m. D.M., or 3·0 to 4·6 p.p.m., if young plants, which tend to have a relatively high copper content even on copper deficient soil, are excluded. The copper content of capeweed on areas known to be " sound " ranged from 7·6 to 13·5 p.p.m. D.M. Samples of capeweed from calcareous soils in South Australia gave values ranging from 2·0 to 3·3 p.p.m. D.M., with a value of 4·9 for the smaller plants, while 3 samples from " sound "

areas in South Australia gave values of 9·3, 11·8 and 12·8 p.p.m. D.M. Although these results indicate good correlation between "soundness" and copper content of capeweed, Beck points out that entirely different results have been reported by Teakle *et al.* (1941). Beck states that, at Gingin, a season favouring ataxia is usually one in which there is a rank growth of capeweed. Sheep grazing on such pastures show bad scouring, which may upset the absorption of copper. The copper content of 2 samples of lupin seeds from the Gingin ataxic area were 5·0 and 7·9 p.p.m. D.M. Bennetts and Beck (1942) point out that the relatively high copper content of the seeds of lupins, which grow well in parts of the Cretaceous area, may be important in relation to the improvement in the haemoglobin values of ewes during the summer months, but the amount obtained then is inadequate to prevent the occurrence of ataxia in the progeny some months later.

Analyses of samples of mixed pasture taken throughout the growing season from the same area showed the highest copper content in the very short pasture. In general, there was a fall in copper content as the plant approached maturity; e.g. at Gingin, pasture which had 3·1 p.p.m. D.M. of copper at the very short stage had only 1·3 p.p.m. at the later dry stage. In practice this seasonal variation is complicated by the effect of close grazing which appears to reduce the copper content of the pasture. It is assumed that the sheep selectively graze the leafy material leaving the stalks and stems of lower copper content (Beck, 1941 [3]).

Beck (1941 [3]) correlated his results for the copper content of West Australian mixed grass and clover pastures with the "soundness" of the areas for stock, his criteria of soundness being: no ataxia in lambs or "falling disease" in cattle, no stringy wool in sheep, stock "do well." He tentatively suggests that values of less than 3 p.p.m. D.M. indicate unsound land, values between 3 and 5 are marginal and values over 5 p.p.m. indicate sound land. He points out, however, that one or more factors appear to operate in certain seasons to influence the availability of the plant copper to animals. Some support is lent to the hypothesis that the copper in rank green pastures is less available by the fact that although the copper content of the pasture in the dry summer state is lower than in winter and spring the symptoms of deficiency are less marked in the summer months. The high incidence of ataxia observed by Bennetts and Beck (1942) in untreated lambs at Gingin in 1937 compared with 1938 and 1939 could not be explained as due to differences in the copper content of the pasture. Possible explanations were the contamination of the pasture by the faeces of sheep given copper drenches or licks in previous seasons or the concentration of copper in small areas, e.g. sites of old camps, but it is considered possible that seasonal differences were predominantly responsible. In 1937 the early winter rainfall was above normal and dull days were frequent; the 1938 and 1939 seasons were unusually dry and sunny. It is stated that these seasonal differences were associated with differences in plant growth and in species distribution in the pasture, but no details are given. Stock owners believe that seasons which favour a luxuriant growth of pasture favour the occurrence of the disease, this being especially so when there is a rank growth of capeweed (see above). The higher incidence of ataxia in seasons favouring good growth of pasture has also been remarked upon in South Australia (Marston *et al.*, 1938).

The copper content of grass and clover pasture growing on the faintly acidic coarse red sand at Gingin was increased from 2·5 p.p.m. D.M. to 14·8 p.p.m. about 3 months after being topdressed with 20 lb. bluestone to the acre, and the increase persisted, although not at the same level, for at least 3 seasons. This result is in marked contrast to results obtained in South Australia where the application of 1 cwt. bluestone per acre caused only a relatively small increase in the copper content of pasture species growing on highly calcareous soil. Apparently the *p*H of this soil prevents proper absorption (Beck, 1941 [3]). According to Bennetts and Beck (1942) topdressing "affected" pasture with copper sulphate is an effective method of controlling ataxia in Western Australia.

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"SWAYBACK" IN LAMBS. UNITED KINGDOM

Occurrence and Description of the Disease

A disease in lambs known locally by a variety of names such as "swayback" and "swingback" appears to have a wide geographical distribution throughout England, Wales and Scotland. Innes and Shearer (1940) state that they have personally investigated or received cases from 15 English and 6 Welsh counties. Roberts (1938) has found a similar wide distribution. The disease has also been reported in other English counties, and Roberts has recorded the occurrence of "swayback" in widely separated districts in Scotland. Investigations into the etiology of "swayback" seem to have been confined to North Derbyshire, where, according to Dunlop and Wells (1938), the disease is endemic over an area of 25,000 acres on soils overlying limestone, gritstone and limestone shale formations. The affected area is irregularly traversed by old lead mines which have become covered with pasture, but the disease occurs in this area and in other areas of England where there are no known outcrops of lead (Innes and Shearer, 1940).

"Swayback" is a nervous disorder of newborn and young lambs first described by Stewart (1932) and said to be similar to diseases of lambs reported in South America, Sweden, South Africa and India (see p. 46), and to enzootic ataxia in Australia. The symptoms are spastic paralysis of the limbs with resultant inco-ordination and occasionally blindness. There is no loss of appetite. In most cases the disease progresses to a fatal end, but mild cases are found in which the lambs recover. The longer the period elapsing between birth and the onset of symptoms the milder the case. The incidence varies from year to year in the same district, and ewes may produce alternately healthy and affected lambs in successive seasons on the same farm. All breeds seem to be equally susceptible. The pathology is characterised by lesions of the white matter of the brain. Variations in the severity of the lesions, not correlated with the age of the affected animals, probably indicate that acute and chronic types of the disease occur. Degeneration of the spinal cord is also characteristic.

In areas of Derbyshire where "swayback" in lambs is enzootic, pregnant ewes are frequently anaemic, but there is no correlation between the anaemia in the ewes and the occurrence of "swayback" in the lambs (Innes and Shearer, 1940). No abnormality was found on post-mortem examinations of ewes giving birth to "swayback" lambs.

Ewes are more prone to give birth to "swayback" lambs when they are pastured on "affected" land for more than one season. The disease is said to be prevented by transference of ewes to unaffected areas during pregnancy, from about November until lambing time. If large amounts of hay and concentrates have to be fed to the pregnant ewes on account of a severe winter, the incidence is said to be reduced.

Experimental Studies

Innes (1934–35), on the basis of negative findings in bacteriological investigations and inability to transmit the disease experimentally to healthy lambs, concluded that neither bacterial nor virus infection was concerned in the etiology of "swayback." This opinion was shared by Dunlop and Wells (1938) on the basis of general observations.

Feeding experiments. Dunlop and Wells (1938), in a large scale experiment with 1800 ewes on 51 farms where "swayback" was endemic, demonstrated that feeding licks containing 0·3 per cent. copper to ewes during gestation reduced the incidence of "swayback" in the progeny. Licks containing cobalt or manganese but without copper were ineffective. The percentages of affected lambs in the different groups were: control 15·2, manganese 15·6, cobalt 10·3, cobalt, manganese and copper 1·97, copper 1·75. Owing to practical difficulties only one type of treatment was allotted to any one farm. In a later experiment (Dunlop *et al.*, 1939) in a "swayback" area the effect of giving licks containing 0·3 and 1 per cent. copper as copper sulphate for periods ranging from 12 to 4 weeks before lambing was observed in pregnant ewes. Ewes fed 1 per cent. copper for 10 or 12 weeks before lambing produced no "swayback" lamb out of a total of 43 lambs born, and only 2 in 34 lambs developed "swayback," when 1 per cent. copper was given to the mothers for 7 or 4 weeks before lambing. The occurrence of "swayback" in lambs was also prevented when licks containing 0·3 per cent. copper were given to ewes for 12 weeks before lambing. Reduction in the period of treatment increased the incidence of "swayback" and with 4 weeks' treatment 8 lambs out of 28 were affected. In a control group of ewes which produced 123 lambs, 36 developed "swayback." Throughout these experiments supplementary feeding was needed because the ground was covered with snow most of the time. This

consisted of hay from " affected " farms, concentrates and sugar beet pulp. The copper content of the mixed rations ranged from 6·5 to 10·5 p.p.m. during the experiment.

Composition of the blood. Dunlop and Wells (1938) observed that the ewes in " swayback " areas of Derbyshire were frequently anaemic. In a detailed haematological study Innes and Shearer (1940) found, in general, a lower level of haemoglobin and red cell count of pregnant ewes in Derbyshire, where " swayback " is enzootic, than among control ewes in Cambridgeshire. There was no correlation, however, between the anaemia of the ewes and the occurrence of " swayback " in the lambs. Some ewes giving birth to affected lambs had blood pictures within normal limits, and others giving birth to normal lambs had haemoglobin values as low as 44 per cent. (Hellige) and red cell counts as low as 3 million per c.mm. Some, but not all, of the Derbyshire pregnant ewes showed high colour indices and some showed morphological changes in the erythrocytes. A similar lack of uniformity was observed among the lambs. Many severe cases of " swayback " showed values for haemoglobin and red cell counts within the normal range, while others showed a reduction in both haemoglobin and red cell counts.

Innes (1934–35) determined the calcium, inorganic phosphorus and magnesium in the blood of " swayback " lambs and their mothers. Although the majority of the values appeared to be normal, a number of the lambs and their mothers showed low values for blood phosphorus. The significance, if any, of these findings is not apparent. Innes and Shearer (1940) found that the blood copper of " swayback " lambs was much lower than that of healthy lambs in non-affected areas. The copper content of the mothers' blood was also low and there seemed to be some relationship between the blood copper of the lamb and its mother. Low values were also obtained for the blood copper of ewes on " swayback " farms giving birth to normal lambs, and it appears, therefore, that the blood copper level of the mother does not determine the development of " swayback " in the offspring. The results are presented in Table 1.

TABLE 1

THE COPPER CONTENT OF THE BLOOD OF EWES AND LAMBS FROM " SWAYBACK " AND HEALTHY AREAS

Animals		Blood Cu: mg. per 100 c.c.	
Description	No.	Range	Mean
" Swayback " lambs, Derbyshire	22	0·024–0·084	0·055
Normal lambs, Cambridgeshire	8	0·057–0·379	0·177
Mothers of " swayback " lambs, Derbyshire	15	0·037–0·084	0·058
" " " Gloucestershire	8	0·046–0·103	0·069
Ewes on " swayback " farms giving birth to normal lambs	19	0·034–0·061	0·045
Normal pregnant ewes from non-affected areas			
Cambridgeshire	17	0·054–0·281	0·127
Leicestershire	5	0·047–0·127	0·073
Derby (non-affected area)	4	0·082–0·122	0·095

These findings were confirmed by Shearer *et al.* (1940) who made analyses of the blood of the experimental sheep described in a previous report (Dunlop *et al.*, 1939). It was also observed that the blood copper of the experimental ewes in Derbyshire increased with the advance of pregnancy, the increase being more marked in ewes in which a copper supplement prevented the production of " swayback " lambs than in ewes receiving no supplementary copper or an amount insufficient to control the disease. Eden (1941) studied the blood copper levels of apparently normal Northumbrian sheep and found values for 77 out of 306 sheep in 1939, and 37 out of 118 sheep in the following year below the upper level reported by Innes and Shearer (1940) for ewes giving birth to " swayback " lambs, although " swayback " was unknown on the Northumbrian farms concerned. He concluded that this gave general support to the statement of Innes and Shearer (1940) that the level of copper in the blood of the ewe does not determine the occurrence of " swayback " in the offspring. Shearer *et al.* (1940) found the mean lead content of the blood of 8 affected lambs to be 0·024 mg. per cent. compared with 0·058 for 6 unaffected lambs in the area. The mean value for 6 healthy lambs in Cambridgeshire was 0·024 mg. per cent. In 6 ewes which gave birth to " swayback " lambs the mean lead content of the blood was 0·022 compared with 0·019 mg. per cent. for 6 ewes which produced healthy lambs.

Composition of organs. Shearer *et al.* (1940) and Innes and Shearer (1940) have shown that lambs and their mothers in affected areas in Derbyshire have liver copper contents lower than are

found in healthy areas elsewhere, irrespective of whether the lambs are affected with "swayback" or not. The copper reserves in unaffected lambs, however, were somewhat higher than in affected lambs. Analysis of the brains of lambs and ewes for copper showed no difference between affected and unaffected lambs or between the corresponding groups of mothers.

The lead content of the liver, brain and ribs of lambs and their mothers in affected areas was uniformly high whether or not the lambs were affected with "swayback." The results of Shearer *et al.* (1940) for the mean copper and lead contents of tissues are presented in Table 2.

TABLE 2

THE COPPER AND LEAD CONTENT OF TISSUES OF EWES AND LAMBS IN "SWAYBACK" AREAS

Tissues	Cu: mg. per cent.		Pb: mg. per cent.	
	Unaffected	Affected	Unaffected	Affected
Lambs				
Liver	1·84 (31)	1·48 (13)	2·3 (35)	1·2 (11)
Liver from normal lambs (Cunningham, 1931)	11			
Brain	0·75 (30)	0·84 (9)	0·16 (29)	0·22 (7)
Ribs	—	—	0·94 (9)	1·17 (9)
Ewes				
Liver	1·6 (8)	1·93 (2)	0·7 (4)	2·4 (2)
Liver from ewes in healthy area (Cunningham, 1931)	32			
Brain	0·73 (5)	0·71 (2)	0·14 (5)	0·11 (2)
Ribs	—	—	3·6 (2)	2·9 (2)

Values for livers and brain are expressed as mg. per cent. of dry tissue and for ribs as mg. per cent. of fresh bone. Figures in brackets refer to the number of animals examined.

Shearer *et al.* (1940) point out that any toxic effect of lead in the ewes could be of only a very chronic nature, but there is no evidence to indicate the occurrence of even this insidious form of plumbism. They conclude that lead does not play a major role in the etiology of the disease. Since reduction of the copper reserves in the liver or in the blood of the ewe does not occur to an extent sufficient to cause recognisable pathological effects in the ewe, they suggest that the problem may be one of transference of copper from the mother to the foetus.

Composition of the Pasture

Chemical analyses of pasture from "swayback" farms in Derbyshire, control farms near this swayback" area, "swayback" farms in Gloucestershire and from healthy control farms away from the "swayback" areas were made by Innes and Shearer (1940) (see Table 3). The copper content of the 15 samples analysed from widely separated areas ranged from 1·2 to 2·7 mg. per 100 g. D.M. with no difference between "swayback" and control areas. The lead content showed a wider range of values. Grass from "swayback" farms in Derbyshire, except in one instance, showed a lead content of over 5 mg. per 100 g. D.M. with one high value of 39·6 mg. per 100 g. The lead content of pasture from control farms in or near the "swayback" areas in Derbyshire ranged from 4·4 to 8·5 mg. per 100 g. D.M. Samples from "swayback" farms in Gloucester gave 1·9 and 11·9 mg. per 100 g., while the lead content of the control farms away from "swayback" areas was less than 0·05 mg. per 100 g. The amounts of iron in the grass from both "swayback" and control farms in Derbyshire and also from "swayback" farms in Gloucester were much higher than is usually found in grass.

In respect of the other constituents except calcium, the grass from affected farms compared unfavourably in most instances with that of good pasture, although no gross deficiency of any constituent was evident. The high calcium to phosphorus ratio of many samples of grass from North Derbyshire was later commented upon by Shearer *et al.* (1940). They also stated that spectroscopic examination of samples of pasture from affected areas in Derbyshire showed normal amounts of trace elements, including cobalt, and high amounts of lead and zinc, and suggested the possibility that the high calcium to phosphorus ratio, the high lead or the high zinc of the pasture may interfere with the absorption of copper from the alimentary tract.

TABLE 3

MINERAL CONTENT OF GRASSES (per cent. D.M.)

Type of Farms	No. of Farms	Total Ash	CaO	P_2O_5	$\frac{CaO}{P_2O_5}$	Fe	Cu mg.	Pb mg.
" Swayback " farms (Derbyshire)	6	9·84–14·43 (12·93)	0·63–2·39 (1·20)	0·38–0·67 (0·54)	2·2 : 1	0·04–0·16 (0·12)	1·4–2·7 (2·0)	0·9–39·6 (11·6)
Control farms in or near " swayback " areas	3	8·54–10·71 (9·80)	0·30–1·3 (0·82)	0·49–0·78 (0·60)	1·4 : 1	0·07–0·14 (0·11)	1·5–1·9 (1·7)	4·4–8·5 (6·6)
" Swayback " farms (Gloucestershire)	2	9·99, 9·95 (9·97)	0·52, 0·72 (0·62)	0·56, 0·68 (0·62)	1 : 1	0·10, 0·09 (0·10)	1·9, 1·7 (1·8)	11·9, 1·9 (6·9)
Control farm (Northamptonshire)	1	23·33	2·24	0·74	3 : 1	0·02	2·4	0·5
Control farms away from " swayback " areas	3	11·8, 10·12 (10·96)	0·79, 0·85 (0·82)	0·91, 0·66 (0·78)	1 : 1	0·04–0·05 (0·05)	1·2–1·8 (1·5)	< 0·05 < 0·05

Shearer *et al.* (1940) analysed samples of pasture from 5 experimental fields in the " swayback " area of North Derbyshire in November and in January. The copper and lead of the grass increased from November to January, but lead to a much greater extent than copper. The values for copper ranged from 0·8 to 1·3 mg. per 100 g. D.M. in November and from 0·8 to 1·8 mg. per 100 g. in January. The corresponding values for lead were 3·7 to 7·0 in November and 8·5 to 35·2 mg. per 100 g. D.M. in January.

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" SWAYBACK " IN OTHER COUNTRIES

A condition said to resemble " swayback " in Britain has been reported in lambs in Peru, Patagonia, Argentina (Gaiger, 1917), Sweden (Magnusson, 1920), India (Krishnappa, 1936), South Africa (Dunning, 1933) and New Zealand (personal communication from Hopkirk to Innes and Shearer, 1940), but no comparable disorder has been observed in Germany (Innes and Shearer, 1940). Innes and Shearer (1940) conclude that, in spite of the absence of accurate pathological data, the similarity of the history, symptoms and age incidence of affected lambs might indicate that the condition in these countries is comparable to " swayback " in Britain. According to Innes (1943) a recent publication by Tabusso (1941–42) leaves no doubt of the identity of " renguera " in Peru and " swayback " in Britain. In Peru it has been suggested that the incidence of the disease in lambs was associated with contamination of pastures with lead from the large number of blast furnaces in " affected " areas, but apparently in 1916–22 when the incidence of the disease was declining the blast furnaces were working at full production. Good results have been achieved by administering copper supplements to the ewes.

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" SUDDEN DEATH " OR " FALLING DISEASE." AUSTRALIA

OCCURRENCE AND DESCRIPTION OF THE DISEASE

An enzootic disease of dairy cattle characterised by sudden death was first brought to the notice of the Department of Agriculture, West Australia, in 1929. The area affected is a belt of country adjacent to the coast in the south-western corner of West Australia, known as Busselton, bounded on the west coast by Wonnerup and Forrest Grove and extending inland for a maximum of 10 miles.

On the south coast the disease appears to be confined to Northcliffe on an area 10 to 20 miles from the coast (Bennetts and Hall, 1939). Cases of what appears to be the same disease have been reported in cattle at Woolca-Woolca, near Gingin, and on some other properties affected with coast disease (Bennetts, 1940). The disease has also been reported from King Island (Dumaresq, 1939). In the affected areas at Busselton and Northcliffe, soil types vary considerably, including heavy loam, gravelly soils and grey sand soils ; there is usually a yellow clay subsoil. The coastal fringe of Busselton, which is relatively free from the disease, consists of aeolian sands, coastal limestone and sedimentary deposits around rivers and estuaries (Bennetts and Hall, 1939).

In areas of South-western Australia, where " sudden death " or " falling disease " is enzootic, clinical symptoms in adult cattle are evident, especially during the flush growth of pasture from August to October when, despite this, there is some loss of condition, a rough, staring coat and evidence of anaemia. Depraved appetite is common. According to Bennetts *et al.* (1941) the syndrome somewhat resembles that described by Sjollema (1938) and Brouwer (1938) in Holland (see p. 50), but diarrhoea, although sometimes present, cannot be regarded as characteristic of the disease in Australia. Suppression of oestrus and temporary sterility in cows and stunted growth and limb abnormalities in young calves are observed. The incidence and distribution of " sudden death " in cows varies from year to year. Deaths may occur from July to December, but the greatest mortality occurs during September and October. From 5 to 40 per cent. of a herd may succumb. Cattle only are affected, but the occurrence of ataxia in lambs has been confirmed pathologically in an area of " falling disease," and conditions in foals and calves similar to those observed in West Australia (see p. 49) occur. In cattle which die of this disease the most striking and constant features are congestion of the abomasum and small intestine, congestion and petechiation of the trachea, a very friable liver and a large, dark, pulpy spleen. The outstanding constant microscopic findings are a characteristic glomerulo-nephritis and haemosiderosis of the spleen and usually also of the liver and kidney. Haemosiderosis is not of general occurrence in dairy cattle in the district apart from " falling disease " cases. According to Bennetts *et al.* (1942 [1]) the primary lesion is atrophy of the myocardium.

During the season when the disease occurs, many cows in affected herds are severely anaemic, but a progressive improvement appears towards the end of the season. The anaemia, which seems to be of the macrocytic hypochromic type, is described in more detail below.

Experimental Studies

Feeding experiments. It is stated that under field conditions the widespread use of mineral supplements containing copper has resulted in a marked improvement in the health and production of entire herds, and apparently in the complete prevention of " falling disease " in cows (Bennetts *et al.*, 1941). Recently the effect of copper in preventing the occurrence of " falling disease " has been demonstrated in controlled experiments (Bennetts *et al.*, 1942 [1]).

Composition of the blood. According to Bennetts and Hall (1939) and Bennetts *et al.* (1941) values as low as 4·6 g. haemoglobin per 100 c.c. and red cell counts of 2·4 million per c.mm. are commonly observed between August and October. Mean cell volumes ranging from 73 to 113 (mean 88) c.μ for 7 anaemic cows compared with 47 to 50 (mean 50) c.μ for 7 healthy cows from sound areas are reported by Bennetts and Hall (1939). Data given by Bennetts *et al.* (1941) for the blood picture of 12 anaemic cows from " falling disease " areas and 10 or more healthy cows from sound areas are shown in Table 1.

TABLE 1

The Blood Picture of Cows from " Falling Disease " and Healthy Areas

	Hb g. per cent.	R.B.C. mill. per c.mm.	Cell Vol. per cent.	Mean Cell Vol. c.μ.	Colour: Count Ratio	Colour: Volume Ratio
Healthy cows . . .	11·0–16·7 (13·3)	5·6–9·4 (7·2)	30·5–46·3 (36·1)	46·7–55·9 (50·6)	1·7–2·1 (1·86)	0·36–0·41 (0·37)
Anaemic cows . . .	4·6–7·4 (6·7)	2·0–3·6 (2·8)	17·5–28·8 (23·5)	66·7–120·0 (86·7)	1·9–3·0 (2·5)	0·25–0·38 (0·29)

They concluded that the anaemia is of the macrocytic hypochromic type.

Bennetts and Hall (1939) prevented anaemia by administering a mineral supplement of iron, copper, cobalt, zinc, manganese and nickel to cows in a " falling disease " area where several samples of pasture contained less than 2 p.p.m. D.M. of copper. Anaemia developed in the control half of the

herd which did not receive any supplement, but no case of " falling disease " occurred in the herd that year (1938). Bennetts *et al.* (1941) found that a supplement of copper alone prevented anaemia and kept cows in apparently optimal health. The addition of cobalt, zinc, manganese, nickel and iron did not appear to confer any additional benefit on cattle treated with copper. The effect of the copper supplement is shown in Table 2.

TABLE 2

The Effect of a Copper Supplement on the Blood of Cows in a " Falling Disease " Area

Supplement		April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
None	Hb g. per 100 c.c.	10·4–11·4 (10·9)	9·2–11·4 (10·4)	8·9–10·6 (9·9)	9·8–11·5 (10·5)	8·6–11·1 (10·1)	4·7–8·9 (5·9)	5·6–10·2 (7·6)	9·0–13·1 (10·3)	9·4–14·0 (11·9)
	R.B.C., pooled sample mill. per c.mm.	—	5·3	5·0	5·6	5·8	2·4	2·4	3·6	5·8
	Blood Cu. : mg. per cent.	0·01–0·04	0·03–0·06	0·02–0·04	0·02–0·04	0·02–0·03	0·01–0·04	< 0·01–0·01	< 0·01	0·01
100 mg. Cu daily from 17th May	Hb g. per 100 c.c.	10·6–15·5 (12·0)	9·6–16·6 (11·6)	9·6–15·8 (11·7)	10·2–15·0 (11·8)	10·1–14·5 (11·4)	8·1–12·2 (10·3)	9·8–16·1 (12·0)	11·9–16·3 (13·6)	13·5–16·5 (15·3)
	R.B.C., pooled sample mill. per c.mm.	—	6·0	5·5	6·4	5·9	4·4	5·3	5·1	6·8
	Blood Cu : mg. per cent.	0·03–0·05	0·03–0·07	0·03–0·06	0·06–0·08	0·06–0·08	0·06–0·10	0·08–0·09	0·06–0·07	0·06–0·08

It will be seen that the supplement of copper prevented anaemia and maintained good blood copper levels. In the control group an improvement in haemoglobin and red cell count which took place in December was not accompanied by a corresponding improvement in blood copper level. No case of " falling disease " occurred in the experimental herd. Beck (1942 [2]) points out that, although the correlation between low blood copper and the development of anaemia in cows grazing on pasture containing 1 to 3 p.p.m. D.M. of copper is not perfect, anaemia usually occurs if the blood copper level is below 0·01 to 0·02 mg. per cent. for any length of time. He states that a rise in blood copper generally takes place in the late spring in affected areas although no seasonal variation was observed in " sound " areas. The blood copper levels of healthy cows in " sound " areas ranged from 0·07 to 0·17 mg. per cent. with haemoglobin values of 8·6 to 16·8 g. per 100 c.c.

Composition of organs. Bennetts *et al.* (1941) also analysed the livers of cattle in " affected " and " sound " areas for copper and found relatively low values in the former (Table 3).

TABLE 3

The Copper Content of the Liver in Healthy and " Falling Disease " Areas

Cattle : Description	No.	Liver Cu : p.p.m. D.M.	
		Range	Mean
Adult cows dead of " falling disease " . .	6	1 to 6	2·1
Adult cows 4 Yearling 1 } in " affected " areas . . . Calves 5	10	1 to 5	3·7
Healthy cows from " sound " areas . .	6	37 to 231	122

Composition of cows' milk. Beck (1941 [1]) found that values for the copper contents of milk from cows in affected areas where the pasture contained 1 to 3 p.p.m. D.M. of copper were low, mostly 0·01 to 0·02 mg. per litre, compared with values for cows in sound areas where the majority lay between 0·05 and 0·20 mg. per litre.

Bennetts *et al.* (1942 [1]) have recently confirmed their earlier findings on the haemoglobin and copper status of animals in " falling disease " areas and the response to copper supplements. They conclude that " falling disease " is the terminal phase of an acute copper deficiency accompanied by a transient anaemia.

Composition of the Pasture

The holdings in the Busselton and Northcliffe districts were originally sown with subterranean clover (*Trifolium subterraneum*). This did well initially, and in some places still persists ; in others this clover has " stalled " and its place has been taken by drooping flowered clover (*T. cernuum*). Almost without exception " falling disease " has occurred on areas which have reverted to drooping flowered clover. In general, during the season of the disease, the pasture appears to be predominantly a luxuriant growth of drooping flowered clover associated with subterranean clover. Grasses come into prominence later in the year, and deaths become infrequent or cease with the predominance of grass over clover. The following are then commonly observed : yorkshire fog, sweet vernal, silver grass, spear grass, ryegrass, barley grass. Capeweed, flatweed and sorrel are also common. On the wetter portions Lotus major, paspalum and kikuyu grow luxuriantly and pastures may be green throughout the summer. The pastures are topdressed annually with 1 to 2 cwt. of superphosphate per acre, without which treatment they revert to yorkshire fog, hop clover and the above-mentioned weeds. The possible relationship of the disease to the high proportion of *T. cernuum* was not overlooked, but preliminary determinations of the copper status of animals and pasture indicated a copper deficiency, and the reversion to *T. cernuum* appears to be associated with the different copper requirements of the 2 species (see p. 41), (Bennetts and Hall, 1939).

Beck (1941 [3]) found that samples of mixed clover-grass pasture from properties on which " falling disease " had been reported showed a copper content of 1·1 to 2·2 p.p.m. D.M. and usually less than 2 p.p.m. Other mixed samples from nearby properties where the disease had not occurred had copper contents of 1·1 to 8·0 p.p.m. D.M. The absence of the disease on properties with low copper content is attributed to the provision of copper salt licks for cows. Most of the healthy pasture samples contained between 7 and 12 p.p.m. of copper. Samples of capeweed, excluding small plants, from properties where " falling disease " had occurred gave values of 1·3, 2·9 and 4·2 p.p.m. D.M. of copper compared with values of 7·6 to 13·5 for areas known to be sound. According to Bennetts *et al.* (1942 [2]) Dumaresq has demonstrated a low copper content of pasture on King Island where " falling disease " occurs.

Elliott (1939) has shown that topdressing with copper compounds has improved pastures on affected properties, notably in regard to the re-establishment of subterranean clover.

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SO-CALLED RICKETS OR " WOBBLES " IN FOALS AND CALVES AUSTRALIA AND U.S.A.

Occurrence and Description of the Disease

A condition known locally as " rickets " occurs in foals and calves in the areas of Australia where ataxia is enzootic in lambs and " falling disease " occurs in cattle. A similar condition has been reported in foals in parts of the U.S.A., where it occurs sporadically on limestone areas or on soils overlying limestone.

The condition in foals in Australia has been described by Bennetts (1935). It is characterised by unthriftiness, depressed growth rate, and in the more severe cases by pronounced stiffness of the limbs and enlargement of the joints. Affected animals frequently stand upon their toes, apparently as a result of contraction of the tendons. The condition may be apparent at birth, or may develop within the succeeding 6 months, but does not develop after weaning although foals tend to remain unthrifty and poorly grown until 2 years old, even when grazing excellent pasture and fed concentrates. Bennetts and Beck (1942) point out that the essential clinical features in foals and calves are malnutrition and abnormalities in structure and posture of the limbs ; they say that there is never

any indication of ataxia or any sign of involvement of the central nervous system, although the pathology has been studied only incompletely. Olafson (1942), who studied " wobbles " in foals in America, drew attention to the similarity of the symptoms, spinal cord lesions and progress of the disease in foals to those in ataxia of lambs. He stated, however, that exercise, which accentuates the symptoms in ataxic lambs, did not make the colts worse.

According to Bennetts (1935) the disease in foals can be prevented or its progress arrested by stable feeding the mother with concentrates, and complete and rapid recovery is obtained by transferring mother and foal to " sound " pasture.

Experimental Studies

Feeding experiments. Bennetts (1935) reported excellent results in prevention and cure by feeding ammonium chloride to the mares before parturition and during lactation. It will be remembered, however, that in the case of ataxia, the beneficial effect of this treatment was later attributed to impurities in the ammonium salt, and according to Bennetts and Beck (1942) there are indications that a copper supplement is beneficial to affected foals.

Composition of the blood. Olafson (1942) reported haemoglobin values of 9 and 9·5 g. per 100 c.c. for 2 " wobbler " foals. He also found blood copper levels of 0·05 mg. and 0·17 mg. per 100 c.c. blood respectively, for the dam of a " wobbler " foal and for a foal affected for several months with " wobbler ".

Composition of the Pasture

The low copper content of the pasture in the enzootic ataxia and " falling disease " areas where " rickets " in foals and calves also occurs has already been demonstrated.

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" STRINGY " OR " STRAIGHT STEELY " WOOL IN SHEEP. AUSTRALIA

" Stringy " wool in sheep has been reported in those areas of Australia where animals develop " coast disease," " falling disease," and enzootic ataxia. It occurs also in areas of Western and South Australia where these diseases have not been observed, and recently a marked extension of its occurrence further inland has been recorded (Bennetts, 1932, 1942 ; Bennetts and Beck, 1942 ; *J. Dept. Agric. S. Aust.*, 1941).

After a few months on " affected " pastures sheep show the characteristic wool changes. The wool becomes straight, limp and glassy and loses its bulkiness. " Stringy " wool is most evident in fleeces of the merino breed, and is exhibited by sheep of either sex and of any age past the lamb stage. It is more marked during the green feed period and in " flush " seasons than during the dry feed months or during dry seasons when green feed is not so rank and abundant. A change to healthy country leads to a spectacular improvement.

Bennetts and Beck (1942) and Bennetts (1942) have shown that " stringy " wool can be cured and prevented by feeding supplements of copper.

The evidence that enzootic ataxia and " falling disease " are caused by deficiency of copper and " coast disease " by a dual deficiency of copper and cobalt has already been reviewed. The low copper content of the pastures in the first two areas, where " stringy " wool also occurs, has been clearly demonstrated.

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" SCOURING DISEASE." HOLLAND

Occurrence and Description of the Disease

In the course of investigations on " liksucht " (see p. 54) Sjollema (1938) observed a scouring disease in cattle and goats kept on freely drained sandy soil, and a similar condition has been reported by Brouwer *et al.* (1938) in cattle and goats grazing on the " Polders " or sandy soil reclaimed from the Zuyderzee.

The disease is characterised by severe diarrhoea, loss of weight and depigmentation of the hair in black cattle, although Brouwer *et al.* did not observe any correlation between the depigmentation and the severity of the disease. The resemblance to the scouring which occurs in Somerset was remarked upon, and it was concluded that the condition was the same in these two areas. Brouwer *et al.* also compared the " Polder disease " with wasting diseases such as " liksucht," bushsickness and pining, but observed that in these diseases, although diarrhoea might occur towards the end, it was not, as in " Polder disease," a dominant symptom throughout. Sjollema reported that affected animals lose their appetite but drink a great deal, and both workers observed a reduction in milk yield from affected cows. Brouwer *et al.* found in some cases that haemoglobin was below normal, but Sjollema found no evidence of anaemia. In some Polder areas the disease begins very early in the season and by late summer and autumn the diarrhoea is so common that animals must be stalled or moved to healthy pastures. According to Sjollema the disease occurs chiefly during pasture time and may arise from a few weeks to months after the beginning of grazing. Rain seemed to increase the incidence.

Scouring disease can be controlled by transferring affected animals to healthy pasture or by feeding concentrates from healthy areas.

Experimental Studies

Feeding experiments. Brouwer *et al.* experimented with various combinations of minerals and found that the best results were obtained when copper and cobalt were fed ; the haemoglobin of the blood and the dry matter of the faeces were increased and depigmentation was corrected. Copper alone gave very good results, but not quite so good as copper and cobalt. Cobalt, when given alone, was ineffective. Sjollema also reported that the disease was cured by copper sulphate administration.

Composition of the blood. Sjollema found the blood of affected animals to be low in copper and to show a progressive decrease.

Composition of organs. The livers and spleens of affected animals showed copper values lower than normal and excessive deposition of iron. It is not stated whether the iron was in the form of haemosiderin or not. The manganese contents of the livers of 2 affected goats were similar to values for 2 healthy animals. The zinc content of the liver of diseased goats was higher than normal (Sjollema).

Composition of the hair. The hair of diseased cattle was extremely low in copper, values for 5 animals ranging from 1·8 to 3·4 p.p.m., compared with values of 6·6 to 10·4 for 3 healthy cattle. Values obtained for the manganese content of the hair of diseased cattle were 0·76, 0·27 and 0·4 mg. per kg. compared with a value of 0·2 mg. for one healthy cow (Sjollema).

Composition of the Soil, Pasture and Hay

Soil. Sjollema reported a high content of calcium carbonate and sulphur in affected soil.

Pasture and hay. Brouwer *et al.* analysed pastures for proximate principles, phosphorus, potassium, calcium, sodium, magnesium, chlorine, sulphur, manganese, iron and copper and found no difference between pastures on which diarrhoea did and did not occur. The copper content of the former ranged from 5·1 to 10·5 p.p.m. D.M. and the latter from 3·7 to 9·4 p.p.m. Sjollema's analysis of " affected " grass was as follows : crude protein 25·6, calcium 0·86, phosphorus 0·4, potassium 3·55, sodium 0·17, chlorine 1·37 per cent. D.M. and iron 393 p.p.m. D.M. One sample had a copper content of 7·3 p.p.m. D.M. compared with values of not less than 10 p.p.m. for normal grass. Sjollema also analysed 3 samples of hay from the affected area and obtained copper contents of 3·6, 3·9 and 4·65 p.p.m., and manganese contents of 43, 30 and 28 p.p.m. compared with an average of 170 p.p.m. and a minimum of 45 p.p.m. for the manganese content of 14 healthy samples. The iron contents of 2 samples of affected hay were 121 and 131 p.p.m. The zinc content was low and the lead normal.

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" SCOURING DISEASE " ON " TEART " PASTURES. UNITED KINGDOM

Occurrence and Description of the Disease

In Somerset there is an area estimated by Gimingham (1910) at 20,000 acres in which pasture causes scouring in cattle. This land is known locally as " teart," and similar land occurs in Warwick and Gloucester. A relationship between " teart " pastures and the Lower Lias geological formation was observed by Gimingham (1910) and has been confirmed more recently in Osmond's geological

survey referred to by Ferguson *et al.* (1940). The soil of " teart " areas is stiff, unyielding and close in texture, and draining is said to have a beneficial effect on the incidence of scouring.

Milk cows and young cattle are the principal sufferers, but sheep are also susceptible. The disease may occur as early as May or June. It is frequently present and always at its worst during the months of August, September and October, particularly in mild, damp seasons. The condition in cattle is characterised by severe persistent scouring which may appear on the first day or at any time up to 6 weeks after grazing on " affected " pasture begins, but usually after 8 to 10 days continuous grazing. Affected animals lose weight, develop harsh, staring coats, and their condition steadily deteriorates. Milk yield is affected, and it is said that animals will suffer permanent injury if left on the pasture too long. Depigmentation of the hair occurs ; Red Devon cows turn muddy yellow and black cattle become rusty in colour. Sheep, although not so severely affected as cattle, show mild scouring. Muir (1936), reviewing earlier work, states that although numerous post-mortem examinations have been made parasites have not been found in affected animals nor ova detected in the faeces. The disease can be controlled by moving cattle from " teart " to healthy pasture.

Experimental Studies

" Teartness " has been known for over 100 years in Somerset, and early investigators have at different times attributed the condition to bacteria, parasites, water supply, poisonous herbs, poor drainage and the physical condition of the soil, but the first real contribution was the discovery by Ferguson and his co-workers of an unusually high molybdenum content in " teart " pasture.

Feeding experiments. These workers proceeded to test the effect of feeding molybdenum salts to cattle. In a preliminary experiment (Ferguson *et al.*, 1938) it was found that feeding soluble molybdates at the rate of 1·36 g. (approximately equivalent to that supplied by the amount of " teart " herbage eaten in a day) caused extreme scouring and loss of condition in 3 out of 5 dry cows at Jealott's Hill. In later experiments (1940) cows on winter rations were drenched or fed with 5·145 g. sodium molybdate, which is equivalent to the molybdenum in 30 lb. D.M. of pasture containing 0·015 per cent. molybdenum (2·04 g.) or about 50 per cent. more molybdenum than is found in " teart " herbage. Out of 12 cows tested in the " teart " area, scouring, apparently identical with that caused by " teart " herbage, occurred in 9 cows in 3 to 5 days ; 3 cows did not scour. Drenching 3 in-calf heifers for 7 days in a non-" teart " area had no effect. Of 9 strong beasts drenched at Jealott's Hill 3 scoured and 6 did not. Since the winter rations are normally rather binding, trials were also made on cows grazing on non-" teart " pasture and receiving no cake. In Somerset 3 out of 4 cows scoured in 5 to 10 days when drenched daily with 1·715 g. sodium molybdate. Of 6 cows on double this dose (1·36 g. molybdenum) on alternate days, 5 scoured in 4 to 10 days. At Jealott's Hill drenching 4 cows for 10 days with 1·715 g. sodium molybdate (0·68 g. molybdenum) had no effect, but when the dose was doubled 3 scoured in 3 days. It was concluded that the variation in the results demonstrated individual differences in susceptibility and that cows used to " teart " land may be more susceptible than animals used to " sound " land. Preliminary experiments with 2 sheep are reported in which 0·075 g. sodium molybdate was fed daily in addition to a normal ration of hay, and approximately a quarter of the molybdenum ingested was absorbed into the blood stream and eliminated by the kidneys, 99·1 per cent. of the total molybdenum ingested being recovered in the faeces and urine. In a discussion of the possible mode of action of molybdenum on the animal, 3 possibilities are considered ; the effect may be due directly to the molybdenum absorbed into the blood stream ; the molybdenum may prevent some element essential to health from producing its effect ; or molybdenum may catalyse the biological formation of toxins.

Brouwer (1938) pointed out the similarity between the scouring in Somerset and that which occurs in cattle on the Wieringermeer Polder in Holland where the giving of copper and cobalt is curative and preventive. Ferguson *et al.* (1940) therefore tried the effect of copper, cobalt and iron salts on affected cattle. In preliminary trials, Somerset cows caused to scour by drenching with sodium molybdate were drenched on alternate days with other minerals and with sodium molybdate. One cow given 1 g. of copper sulphate and one given 1 g. of copper sulphate and 0·2 g. of cobalt nitrate were cured after the second dose. Another cow responded slightly to 5 doses of 2 g. of ferric chloride. Extensive field trials showed that drenching or feeding cows with 2 g. of copper sulphate and young stock with 1 g. of copper sulphate per head daily cured and prevented scouring on very " teart " land. They concluded that smaller amounts of copper sulphate would probably be sufficient on mildly " teart " land. The addition of cobalt nitrate apparently did not increase the effect of the copper sulphate.

Composition of the Soil and Pasture

Soil. The association between " teart " pastures and the Lower Lias formation referred to by Gimingham (1910) was investigated by Ferguson *et al.* (1940) who quoted reports by Osmond (1934) of

a soil survey of 75,000 acres in central Somerset comprising 4 soil series derived directly and 4 derived indirectly from the Lower Lias. The surface soils derived directly from the Lower Lias contain relatively large amounts of molybdenum, are calcareous and alkaline in reaction and give rise to very " teart " pastures. The series derived indirectly from the Lower Lias include " teart " and non-" teart " soils. If the surface soil is neutral or alkaline in reaction and is calcareous, the pastures are " teart " unless the molybdenum content of the soil is very low. If the surface soil is acid the pastures are not " teart " even though the molybdenum content of the soil may be fairly high. It can therefore be predicted whether a soil in central Somerset is " teart " or not if the series, the molybdenum content, the *p*H and the calcium carbonate content of the soil are known. In some series which are naturally high in molybdenum and are calcareous it is only necessary to know the series for prediction of " teartness." The molybdenum content of the Lower Lias soils varies from 0·001 per cent. to 0·01 per cent. in the surface horizon, which is equivalent to 20 to 200 lb. molybdenum per acre in the top 9 inches of soil.

Pasture. Gimingham (1910) pointed out that no reliable evidence existed to support the theory commonly advanced that " teartness " was due to the presence in the pasture of one or more particular species of plant such as purging flax or carnation grass. On the contrary, the type of herbage in scouring fields might vary considerably, and " teart " land could not be recognised by inspection of the herbage. Chemical analyses of the pasture threw no light on the problem (no data given). " Teart " pasture which had been frosted or made into hay seemed to lose its " teartness," except in the case of very " teart " pasture when the hay still scoured cattle. According to Muir (1936) opinions differ as to the effect of hay from " teart " land, but most farmers agree that after 1 year in the stack hay from " teart " land does not cause scouring. " Teartness " of pasture and of hay appears to become less with maturity. Ploughing up " teart " pasture and re-seeding has been found to have a temporary beneficial effect.

Ferguson *et al.* (1938, 1940) made chemical analyses of samples of Somerset " teart " pasture and reported that the only peculiarity was in the molybdenum content. They found " teart " herbage to contain 0·002 to 0·01 per cent. D.M. of molybdenum compared with herbage of nearby non-" teart " fields which usually contained less than 0·0005 per cent. The degree of " teartness " varied roughly with the molybdenum content. They found that clover separated from a sample of herbage from a very " teart " field contained 0·0095 per cent. D.M. of molybdenum while the grasses contained only 0·0050 per cent. The uptake of molybdenum by several grasses and clovers from a " teart " soil was studied in a pot experiment. The results confirmed the high molybdenum content of clovers compared with grasses. Among the grasses yorkshire fog showed an outstandingly high molybdenum content but is seldom an important constituent of " teart " pastures.

The copper content of both " teart " and non-" teart " herbage varied from 0·0011 to 0·0018 per cent. D.M., figures which were about twice as high as those quoted by Brouwer (1938) for scouring pasture on the Wieringermeer Polder, and Ferguson *et al.* concluded that " teartness " was not due to a deficiency of copper in the pasture. Even Brouwer's figures for the copper content of pasture were higher than those associated with copper deficiency elsewhere. Hence Ferguson *et al.* state that since, in a private communication to them, Brouwer has reported the absence of molybdenum from older pasture, the scouring diseases in Holland and Somerset must be due to different primary causes.

Ferguson *et al.* (1940) found that, like the degree of " teartness," the molybdenum content of the herbage was highest in autumn. Figures obtained over a period of 3 years for pasture from one very " teart " centre showed that the molybdenum content was 0·0032 to 0·0049 per cent. D.M. in April, increased to 0·0066 to 0·0083 in September and then fell off to 0·0029 in December. In December the herbage which was safe for grazing was mostly dead, very fibrous, and obviously of low nutritive value. It is suggested that molybdenum passes from the leaf to the roots and perhaps is returned by the plants to the soil in winter.

June hay and winter herbage, although non-" teart," contained amounts of molybdenum which, if present in younger grass, might have been expected to cause scouring. Examination of samples taken in 1937 showed that the " teartness " was more closely related to the amount of water-soluble molybdenum than to total molybdenum. About 70 to 80 per cent. of the molybdenum in green grass, about 40 per cent. in hay and only about 10 per cent. of that in the dead winter herbage was soluble in water.

Ferguson *et al.* (1938) reported that there was no apparent correlation between the total or hydrochloric acid-soluble molybdenum of the soil and that of the herbage. They later suggested (1940) that the *p*H of the soil may govern the uptake of molybdenum by the herbage, a view which was further investigated by means of fertiliser trials.

Gimingham (1910) had reported that application of manures to " teart " pastures resulted in an increase of the trouble. Ferguson *et al.* (1940) point out that the fertilisers referred to were probably basic slag and lime since nitrogen fertilisers are rarely applied to " teart " pastures. They found that

applications of 1 cwt. sulphate of ammonia per acre per cut to very "teart" pasture increased the yields of herbage in spring and autumn, and reduced the molybdenum content by $\frac{1}{2}$ to $\frac{2}{3}$ throughout the whole grazing season. The effect was not entirely due to reduction of clovers since the results indicated that the total amount of molybdenum absorbed from the soil was not increased, the molybdenum content of the grasses being reduced in proportion to the increase in yield.

Application of a nitrogen and phosphorus fertiliser at the rate of $1\frac{1}{3}$ cwt. per acre per cut also increased yield and reduced the molybdenum content of pasture but not to the same extent as did sulphate of ammonia. The nitrogen phosphorus fertiliser also caused a considerable reduction in clovers. Basic slag applied at the rate of 10 cwt. per acre increased the yield in the latter part of the season by increasing the growth of clovers. The slag caused only a small increase in molybdenum content of the herbage, but the increased production of very "teart" herbage is a disadvantage. Ferrous sulphate, which reduces soil alkalinity and burns out clovers, was found to reduce the molybdenum content of the herbage to the same extent as does sulphate of ammonia.

"Teart" pasture can be produced artificially by topdressing with molybdates. Sound pasture at Jealott's Hill was dressed with 20 lb. sodium molybdate in January. In May the herbage was found to have a molybdenum content of 0·019 per cent. D.M., and 4 heifers put to graze on it scoured severely in 3 days. After they had recovered on sound pasture 2 of the heifers were returned to the topdressed pasture where they scoured badly in 5 to 6 days. In August, when the molybdenum content of the herbage had dropped to 0·003–0·01 per cent. D.M., the 4 heifers scoured severely after grazing on it for 5 days.

In a recent report Ferguson *et al.* (1943) have confirmed their earlier findings and show that the feeding of copper in the amounts recommended for "teart" pastures has no adverse effect on cattle.

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LICKING DISEASE ATTRIBUTED TO COPPER DEFICIENCY. EUROPE

Investigators of diseases of livestock in Europe are agreed that the symptoms which are collectively termed "licking disease" are due to different causes in different areas. In Holland and Sweden "liksucht" is attributed to deficiency of copper, and in Schleswig Holstein opinion is divided for and against copper deficiency as the cause of "lecksucht." The present section will be confined to the licking disease of Europe attributed to copper deficiency. Licking diseases attributed to other causes are dealt with on pages 56–62.

Holland.

Occurrence and Description of the Disease

The disease occurs in cattle, sheep and goats in the Eastern Provinces of the Netherlands, Drenthe, Overijsel, Gelderland, North Brabant and Limburg. It appears to be confined to the sand and moor soils, and probably none of the lands reclaimed from the sea within the last few decades is free from the disease.

The first symptom appears to be a marked decrease in normal appetite followed by emaciation. Other symptoms are dry coat, enlarged head, hard faeces frequently covered with mucus, a tendency to general pica and sometimes anaemia. In young animals development is retarded, in mature animals sexual activity is absent, and when cows are affected milk yield suffers. The symptoms are most severe in young heifers, although cattle of all ages are affected. Sheep are not usually kept on such land because of their susceptibility to the disease. The disease usually appears first in the winter, probably while the animals are still being stall fed on local hay. When animals are turned out to "affected" pasture they become more emaciated, chronic diarrhoea frequently supervenes in the later stages and the animals finally die in a cachectic state. Brinkman (1932) has, however, stated that the disease occurs at all times of the year contrary to the usually published statements that it

occurs more commonly in winter than in summer. According to Thijn (quoted by Sjollema, 1933) the disease gets worse on moorland in wet weather with increased growth of grass and is better when there is less grass.

The disease can be controlled by persuading animals to eat suitable concentrates or by transferring them to healthy or curative pastures.

Experimental Studies

Feeding experiments. Affected animals treated with copper and iron recovered, and copper sulphate alone was also curative (Sjollema, 1933). The first effect of the copper treatment was an improvement in appetite. This was followed by gain in weight, improvement of the coat and increased sexual activity, and eventually by a return of haemoglobin levels to normal. The copper was given as a solution of copper sulphate and the daily dose ranged from 0·2 to 0·5 g. A daily dose of 1 g. of copper sulphate was considered too high, and copper in the form of a salt lick was not recommended.

Composition of the blood. Sjollema (1933) found haemoglobin values of less than 40 per cent. (Sahli) in heifers and values as high as 50 per cent. (Sahli) for full grown animals, which are approximately equivalent to 7 and 8·6 g. haemoglobin per 100 c.c. blood. He thought that anaemia was probably a late development in the disease although it was possible that two forms of the disease might exist, one with and one without anaemia. The dry matter content of the blood tended to be 4 or 5 per cent. below normal, and this was attributed chiefly to the reduction of haemoglobin since plasma protein was usually normal. The sugar, acetone, urea and calcium, phosphorus, magnesium, sodium and chlorine contents of the blood were also reported by Sjollema to be normal. The iron content of the blood of affected animals was 20 to 25 mg. per 100 c.c. compared with 35 mg. per 100 c.c. for normal heifers.

Composition of the Pasture and Hay

Sjollema analysed samples of affected hay for calcium, magnesium, phosphorus, potassium, sulphur, chlorine, sodium, iron, copper, manganese and protein. Low values for phosphorus were sometimes obtained especially after drought, but there was no evidence of correlation between drought and the occurrence of this pica. Further, there was no sign of stiffness of the hind legs such as is found in aphosphorosis, and blood phosphorus was normal. The sodium content of the hay varied widely from 0·1 to 0·4 per cent., and the manganese content was sometimes very high and sometimes very low. Some low protein values were observed, but there was no correlation with the occurrence of the disease. The potassium content ranged from 1·5 to 2·4 per cent. and the acid-base balance was considered normal. The only abnormality which was correlated with the occurrence of "liksucht" was the low copper content of the affected hay. Values ranged from less than 1 to 5 p.p.m. D.M., usually between 2 and 3 p.p.m., compared with values for hay from healthy areas which ranged from 6 to 12 p.p.m. with an average of 7·5 p.p.m. D.M. and one value of 5 p.p.m. Pasture which was known to be curative had a copper content of 15 to 30 p.p.m. D.M.

Sjollema reports increased copper content in "liksucht" pastures manured with copper sulphate, and points out that copper sulphate fertilising also cures the "reclamation disease" of plants which occurs in "liksucht" areas.

Sweden.

Svanberg and Nydahl (1941), presenting the results of a survey of the copper content of Swedish pastures, states that at Gisselas, where the copper content of 40 samples of hay ranged from 0·5 to 5·2 p.p.m. D.M. with an average of 2 p.p.m., licking disease occasionally occurs and is controlled by feeding molasses containing 14–16 p.p.m. copper. They point out that in the Svalsand area of Gotland the copper content of the hay indicates that animals might suffer from copper deficiency were it not for the practice of feeding supplements such as molasses and bran to prevent aphosphorosis since these supplements are high in copper as well as phosphorus.

Germany.

A licking disease with clinical symptoms similar to the "liksucht" of Holland has been described in Schleswig-Holstein and in the Schwarzwald area.

Experimental Studies

Feeding experiments. Traulsen (1937) obtained beneficial results from feeding a mixture of concentrates. Affected animals were first given molasses and a copper salt lick to restore their appetite so that they would eat the concentrates offered. Schermer (1939) gave copper sulphate at the rate

of 0·1 g. daily in the drinking water without beneficial result, and intravenous administration of copper gave only a transient improvement. Balance experiments by Krzywanek and Brüggemann (1942), showed that a healthy ox fed "lecksucht" hays from Schleswig-Holstein for a period of 9 months was in negative copper balance when the hay fed contained only 2·5 p.p.m. D.M. of copper and in positive balance when the hay contained 4·2 p.p.m.

Composition of the Hay

Traulsen (1937) analysed "affected" and healthy hays for copper. In the Schwarzwald area the copper content of "affected" hays ranged from 0·00 to 1·0 mg. per 100 g. (86 per cent. D.M. basis), with a mean of 0·320, compared with 0·1 to 1·5 for 5 healthy hays with a mean of 0·660. His values for "affected" hays in Schleswig-Holstein affected areas ranged from 0·00 to 1·00 mg., with a mean of 0·379, compared with a range of 0·10 to 0·70 mg. for 5 healthy hays with a mean of 0·320 per 100 g. The disease in animals was associated with the occurrence of "reclamation disease" in grains. Krzywanek and Brüggemann (1939) summarising published analyses of lecksucht and normal Schleswig-Holstein hays give the following mean values for copper content (Table 1).

TABLE 1

Copper Content of "Lecksucht" Hay:

		Cu p.p.m. D.M.
Danckwortt and Hotzel	normal hay	5·1
	"lecksucht" hay	5·0
Krzywanek and Brüggemann . . .	"lecksucht" hay I	6·6
	,, ,, II	2·5
	,, ,, III	4·2
	,, ,, IV	7·0
	,, ,, av.	5·1

Nicolaisen and Seelback (1938) reported that in Schleswig-Holstein the occurrence of both "reclamation disease" in plants and "lecksucht" in cattle was prevented in 2 successive years by topdressing with copper. They point out that this is in agreement with Sjollema's findings in Holland and criticise Traulsen's conclusion that the disease is not due to copper deficiency on the grounds that, in general, Traulsen's values for "affected" hay in the Schwarzwald were much lower than in healthy areas and that Traulsen's results for 19 affected areas in Schleswig-Holstein, which showed a mean copper content higher than the mean for 5 healthy areas, were not a reliable guide because of the extreme variations observed among the values for "affected" hay. Schermer (1939), however, suggested that the beneficial effect of copper fertilising obtained by Nicolaisen and Seelback was due to its effect on some plant constituent other than copper.

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CHAPTER IV

DEFICIENCY OR IMBALANCE OF SODIUM, POTASSIUM AND CHLORINE

LICKING DISEASE OR LECKSUCHT

Discussion

A wasting disease characterised by depraved appetite has long been known in cattle and sometimes in sheep in many parts of Europe. Early work reviewed by Green (1925), Orr (1929), Leskien (1937) and Lührs (1941) on the etiology of the disease was confused by the fact that in areas where aphosphorosis was prevalent it was not recognised that a lecksucht occurred, different from the pica or osteophagia associated with brittle bone. In Sweden a clear distinction is still not made

although Stalförs (1933) has pointed out that the "skravel" or licking disease, which occurs in the Dalarna and surrounding districts, can be distinguished from osteomalacia by the absence of bone changes.

Excluding investigations which obviously deal with osteomalacia and investigations of licking disease attributed to deficiency of copper, it appears that lecksucht has been attributed either to an absolute deficiency of sodium, a low ratio of sodium to potassium or a low content of potassium in the hay from "affected" areas. These views receive little support from the experimental work done in affected areas or from a general consideration of the needs of cattle for these elements.

The earliest investigation of the requirements of cattle for common salt appears to be that of Babcock and Carlyle (1905) who showed that when stall fed cows were not given access to salt they developed an abnormal craving for it within 2 or 3 weeks and, later on, sometimes as much as a year later, there was a complete breakdown in health marked by loss of appetite, a generally haggard appearance, lustreless eyes, a rough coat and a very rapid decline in both liveweight and milk yield. When these events followed calving in a high producing cow the collapse might be sudden and death rapidly ensue. Administration of sodium chloride was curative and preventive, and in one case potassium chloride was also effective, indicating a deficiency of chlorine rather than of sodium. It was concluded from these experiments that the requirement of salt was 0·75 oz. daily for a 1000 lb. cow with an additional 0·3 oz. for every 10 lb. milk produced. This is equivalent to 15 g. sodium and 23 g. chlorine daily for a cow producing 2 gal. milk.

The experiments of Theiler *et al.* (1927) showed that the growth of young heifers from 1 year to adult weight was not affected by a ration providing only 1·5 g. sodium and 5 g. chlorine daily, although there were calving abnormalities. Du Toit *et al.* (1934) found that cows on an intake of 6·7 g. chlorine daily showed no adverse effect until lactation began to draw off more chlorine than was obtained in the food. Milk production was, however, gradually reduced until the amount of chlorine secreted was less than the intake. A similar compensation occurred on a ration containing enough sodium for growth but insufficient for normal milk production. As a result of these experiments and those of Theiler *et al.* (1927) they tentatively suggest that 1·5 g. sodium and 5 g. chlorine daily are sufficient for growth and 11 g. sodium and 14 g. chlorine daily for a cow producing 2 gal. milk. Du Toit *et al.* (1940) calculate that, assuming a daily D.M. consumption of 16 lb., these requirements for growth are met by a ration containing 0·02 per cent. sodium and 0·07 per cent. D.M. of chlorine, and the requirements of a cow producing 2 gal. milk daily by a ration containing 0·15 per cent. sodium and 0·19 per cent. D.M. of chlorine. Larger amounts would be required for higher yielding cows.

The analysis of good British cultivated pasture given by Orr (1929) shows 0·18 per cent. sodium and 0·95 per cent. D.M. of chlorine which, according to Du Toit's reckoning, would provide abundant chlorine and enough sodium for health and growth but not for good milk production. This is consistent with common experience that the provision of a sodium chloride lick is necessary for high milk yield on such pastures.

A pica prevalent among cattle in Nigeria can be cured by administration of sodium chloride although the pasture contains as much as 0·26 per cent. D.M. of sodium, and it has been suggested that the requirements of cattle for sodium chloride are higher in hot, semi-arid countries owing to excessive losses of water and salt in sweat, and to the need to conserve body water (Anderson, 1933).

In South Africa the sodium content of some pastures is extremely low during winter (0·01 per cent. D.M.) and probably inadequate for growth, but in these areas deficiency of protein and phosphorus are the most important factors limiting growth. Many S. African pastures provide insufficient sodium for the production of 2 gal. milk daily. The chlorine requirements for growth are met at all times, but for a few months during the dry season the supply will be insufficient for the production of 2 gal. milk. In Sweden pastures with as little as 0·03 per cent. of sodium are found. These low values are not correlated with the incidence of deficiency disease, but the supply of sodium is obviously inadequate for milk production.

Analyses of hays in lecksucht investigations in Norway and Germany show a supply of sodium adequate for growth although some healthy and "lecksucht" hays may not provide enough sodium for good milk production. The supply of chlorine in these areas appears to be abundant for both growth and milk production.

It cannot be argued from the absence of licking disease on pastures low in sodium that the disease is not due to lack of sodium unless it is known that sodium chloride has not been given as a lick in these areas. The presence of licking disease on pastures well supplied with sodium is more convincing evidence that the disease is not caused by lack of sodium in the pasture.

It has been suggested that a high ratio of potassium to sodium in the ration is harmful to cattle and the occurrence of lecksucht has frequently been attributed to such an imbalance. An examination of the ratios of potassium to sodium in samples of "lecksucht" and normal hay shows that the

ratio for some "lecksucht" hays was only 2 : 1 and in no case did it exceed 11 : 1, which compares favourably with the ratio of 15 : 1 for good cultivated British pasture (Orr, 1929).

The idea that the ratio of potassium to sodium in the ration of cattle was important in the etiology of lecksucht can be traced back to the findings of Bunge (1873), who concluded from 1-day experiments on himself that an excessive intake of potassium salts impoverishes the organism in sodium and chlorine. This conclusion has not been confirmed by Richards *et al.* (1927) in experiments on young growing pigs, but it was found that a ratio of 10 or 11 of potassium to 1 of sodium in the ration decreased assimilation of nitrogen, calcium and phosphorus. Orr (1929) points out that the evil effects would be most evident when rations were deficient in these constituents ; for instance, the pastures on which Zuntz found malnutrition and pica to be associated with a high potassium to sodium ratio were also low in calcium and phosphorus. With the possible exception of Zuntz' results and those of Grünigen, who found that "lecksucht" pasture in Switzerland had a normal calcium and phosphorus, high potassium (6–7 per cent. D.M. of K_2O or more) and low sodium, lecksucht does not seem to be associated with an abnormal potassium to sodium ratio and, conversely, pastures with high potassium to sodium ratios have been reported (Orr, 1929), apparently without the occurrence of lecksucht.

Further, Theiler *et al.* (1927) were unable to produce signs of lecksucht in heifers fed rations with ratios of 20 or 30 parts potassium to 1 of sodium.

Consistent results have not been obtained with administration of sodium salts in the treatment of lecksucht so there is clearly very little evidence to support the view that lecksucht is caused by an absolute or relative deficiency of sodium in the diet.

Du Toit *et al.* (1934), in an experiment with young heifers extended over 2 lactations, were unable to demonstrate symptoms of deficiency on a ration very low in potassium. On the basis of this experiment, and assuming a D.M. consumption of 16 lb. daily, Du Toit *et al.* (1940) calculate that the requirements of growing cattle are met by pasture containing 0·34 per cent. D.M. of potassium and those of a cow producing 2 gal. milk daily by pasture containing 0·45 per cent. D.M. of potassium. Their analyses of South African pastures show that values as low as these may occur on some pastures for one or two months of the year, but they concluded that the probability of a deficiency of potassium in animals grazing South African pastures is extremely remote. Cooper *et al.* (1938), discussing soil potassium in relation to plant potassium, state that the potassium in sandy soils is considered relatively highly available, but on the other hand potassium is more easily leached out of sandy soils than out of clay soils. Neal (1941), however, has pointed out that plant growth will fail before the content of the element in the plant is reduced to a level inadequate for livestock ; if this is so, pasture can never be deficient in potassium. Undoubtedly "lecksucht" hays analysed by Traulsen in Schleswig-Holstein and by Slagsvold in Norway had a lower potassium content than healthy hays analysed by the same workers, but the values obtained were more than twice those indicated by Du Toit as essential for growth and production of 2 gal. milk daily. Further, healthy hays in some areas may contain less potassium than "lecksucht" hays from other areas. Svanberg (1933), discussing Slagsvold's results, points out that if a potassium content of 1 to 1·38 per cent. dry hay is the cause of lecksucht then 25 to 50 per cent. of all Swedish cattle should be suffering from the disease.

Lührs (1941), on the basis of studies of plant physiology and the effect of potassium salts on animal enzyme activity, has suggested that either deficiency or excess of potassium might involve disturbance of carbohydrate and protein metabolism, but in the absence of any conclusive evidence of such deficiency or excess in "lecksucht" hay the analogy is of doubtful value.

Lecksucht is sometimes attributed to excessive use of potash manures and sometimes said to be cured by fertilising with potash, but there seems to be little reliable evidence in support of either view.

It seems equally improbable that absolute deficiency of alkali (Na + K) or sodium to potassium imbalance in the fodder causes lecksucht. It has been suggested that the alkali alkalescence [(Na + K)—(S + Cl)] is important in the etiology of lecksucht and that low or negative values are characteristic of "affected" hay, but this is not confirmed by the analyses reviewed by Krzywanek and Brüggemann, nor does there appear to be any relation between lecksucht and the total alkalinity of the hay.

The presence of abnormal amounts of organic acids in the urine of animals affected with lecksucht has been demonstrated, indicating disturbance of intermediary metabolism, but the cause of this is not understood and the elucidation of the problem must await further investigation.

The negative nature of this discussion is regrettable but it is hoped that it may indicate the need for a new line of attack on the problem of lecksucht.

Occurrence and Description of the Disease

Lecksucht, or licking disease, has been reported from various parts of Germany, principally East Prussia and Schleswig-Holstein, Sweden, France, Switzerland and Norway. It occurs most frequently on moor and sandy soils.

The condition occurs chiefly in cattle and is characterised by loss of appetite (concentrates are refused and there is a preference for coarse fodder), and the development of a craving which results in the eating of rubbish and licking of earth, clothes and fences. Growth, milk yield, and liveweight are affected. Animals become emaciated; the hair is lustreless, the faeces are usually hard and sometimes there is diarrhoea. Anaemia is sometimes reported. The head is enlarged, movement is difficult, the animals become very nervous and finally die of cachexia. *Post mortem* examination shows only evidence of inanition. Cattle appear to be the principal and, according to some investigators, the only sufferers from lecksucht, and young pregnant and lactating cows are most severely affected. In most cases hay has been incriminated as causing the condition, but in other cases the disease has occurred in grazing cattle.

Beneficial results have been obtained by change to healthy pasture or by feeding hay brought from a healthy area. Early cutting of hay is also advocated.

Experimental Studies

Feeding experiments.—Neumann and Reinhardt (1922) fed young calves exclusively on hay from the reclaimed moors in East Prussia where lecksucht is prevalent. They observed a loss of sodium and reduced excretion of iron in the urine, signs of anaemia, decreased red cell count and haemoglobin, and reduced CO_2 binding capacity of the plasma. Although clinical signs of lecksucht were not produced, the blood and urine findings were regarded as preliminary signs.

Beneficial effects from feeding molasses in lecksucht areas have been reported by Schink (1922) and Kossmag (1930) in Germany, by Slagsvold (1933) in Norway, by Svanberg (1936), and by Svanberg and Ekelund (1943), who also found sugar administration beneficial in Sweden. Grünigen (1935) found that feeding sodium chloride gave temporary relief to affected cattle in Switzerland. Svanberg (1936) found that in North Sweden administration of sodium phosphate was only successful in some areas probably by correcting a deficiency of phosphorus. In Norway, according to Slagsvold (1933), licking disease can be controlled by feeding molasses but not by feeding sugar. Wood ash is also effective, and the beneficial effect of these two supplements is thought to be due to their high content of potassium. Seaweed was also reported to be beneficial, and the disease can be controlled by feeding alkali carbonates, potassium lactate, potassium citrate or sodium salts, especially with lime. In a study of preference in lecksucht cattle in Schleswig-Holstein for various salt licks Schermer (1939) found that a lick containing sodium and calcium was preferred to a mixture of trace elements. The sodium-calcium lick corrected the acidosis which Schermer had observed but continuation with this lick did not prove beneficial. Several earlier workers reported improvement or recovery in lecksucht cattle in response to injection of calcium bound to an organic acid (Meyrowitz, 1922; Neumann and Reinhardt, 1923; Westermann, 1925).

Kirsch (1934) has reported good results from feeding oats and barley straw in East Prussia, and Traulsen (1937) found that pica in Schleswig-Holstein could be cured by feeding a mixture of concentrates, appetite being first of all restored by feeding molasses and a copper salt lick. Schermer (1939) reports that feeding oats and yeast cured Schleswig-Holstein pica. The curative fraction of the oats was in an alcoholic extract and not in the residue or in an aqueous extract. Schermer also obtained a transient improvement by injecting Cuprosol intravenously but the improvement was not maintained.

The experimental work of Theiler *et al.* (1927) is of interest since they did not find any sign of lecksucht in heifers reared from 1 year old to adult weight on rations low in sodium and chlorine with a $K_2O : Na_2O$ ratio of 12 : 1; on rations low in sodium and high in chlorine with $K_2O : Na_2O$ ratios of 20 : 1 and 30 : 1, or on a ration low in potassium.

Recently Krzywanek and Brüggemann (1941, 1942) made digestibility trials and balance studies on a 2 year old ox suffering from lecksucht, fed exclusively on Schleswig-Holstein hay which causes pica, and on a healthy ox fed this "affected" hay. The results showed that with the sick animal during prolonged feeding on faulty hay the utilisation of the organic food constituents became poorer as their digestibility coefficients fell, and at the same time there was a marked disturbance in the mineral metabolism. These workers concluded that the disease was the result of a complex disturbance of the metabolic processes and could not be attributed to a deficiency of any one food constituent. They further suggested that with a faulty diet of this type the synthesis of vitamins of the B complex in the rumen may be impaired so that indirectly a deficiency of one or more

members of the B complex may arise. It should be pointed out, however, that the sick ox showed a marked reduction in food consumption during the experiment and this alone could account for the general disturbance in mineral metabolism observed without giving any explanation of the cause of the loss of appetite.

In the case of the healthy ox there was no loss of appetite during the 9 months of the experiment. The digestibility trials showed decreased digestibility of the organic constituents of the hay, although not to the same extent as in the case of the sick ox, and it is suggested that if the experiment had been prolonged, equally low values would have been obtained. The 3 mineral balance studies on the healthy ox were made 1, 4 and 9 months from the beginning of the experiment. The balances of calcium, magnesium and phosphorus were all negative in the first period. This was interpreted as meaning that the animal had not adjusted itself to the change in diet since there was progressive improvement later and all 3 balances were positive in the third period. The manganese balance was negative throughout despite a relatively high intake. The iron and copper balances improved throughout the experiment and the copper balance, which was negative in the first two periods, became positive in the last period when a faulty hay with higher iron and copper contents was fed. The sodium, potassium and chlorine balances showed an increase in the second period and a fall in the third period when the sodium and potassium balances were negative. The average composition of the hays used is shown in Table II. Hay I was used in the experiment with the sick ox, hay II in the first two periods of the experiment with the healthy ox, and hay III in the third period of this experiment. It will be seen that the changes in balances between the second and third period of the second experiment roughly follows the change in composition of the hay and it is impossible to draw any conclusion from these results.

Schermer (1939), Krzywanek and Brüggemann (1942) and Krzywanek (1942) also investigated the disturbance in acid base balance accompanying the pica. They found abnormal quantities of organic acids in the urine of sick animals indicating a disturbance of intermediary metabolism. Krzywanek and Brüggemann (1942) point out that acidosis is not always observed, and although Schermer (1939) found acidosis in cattle fed "affected" hay from Schleswig-Holstein, Krzywanek and Brüggemann were unable to produce acidosis with hay from the same area. (Some workers have suggested that lecksucht is caused by deficiency of vitamins or amino-acids but there is no reliable evidence in support of these views.)

Composition of the Hay

In the earlier analyses of hay from lecksucht areas quoted by Orr (1929) low sodium or a low sodium to potassium ratio appears to have been the principal abnormality apart from the low phosphorus in areas where osteomalacia was also prevalent. Low values for sodium and phosphorus have also been reported in the Dalarna, Sweden, associated with high chlorine and normal potassium

TABLE I

Norwegian Hays

	Sample	Per Cent. Dry Hay			Na : K Ratio	Alkali-Alkalinität *
		Na	K	Cl		
Pica-producing hays	1	0·2	1·34	0·5	1 : 7	25·24
	2	0·22	1·38	0·52	1 : 6	25·45
	3	0·20	1·04	0·17	1 : 5	26·74
	4	0·27	1·10	0·38	1 : 4	25·92
	5 (salted)	0·8	1·10	1·24	1 : 1	23·40
"Healthy" hay from clay soil in middle of pica areas	6	0·09	2·1	0·5	1 : 23	34·77
Hay from disease-free areas among pica areas	7	0·47	1·80	0·95	1 : 4	36·55
	8 (salted)	0·88	3·48	0·32	1 : 4	112·57
	9 (salted)	2·10	2·00	0·67	1 : 1	114·25

* Milligram equiv. per 100 g. hay.

(Hansson, 1931). These pastures are not deficient in copper (Svanberg and Nydahl, 1941). Svanberg (1936) found that values as low as 0·03 per cent. Na_2O were not uncommon in hay from North Sweden, but these low values were not correlated with "deficiency disease" in livestock. The K_2O contents of hay from 2 very good farms were 3·68 and 2·06 per cent. compared with 1·4 to 1·5 per cent. for samples of hay associated with "deficiency disease."

TABLE II

COMPOSITION OF HAY (PER CENT. D.M.), KRZYWANEK AND BRÜGGEMANN (1939).

Author	Hays	Crude Prot.	Crude Fat	Crude Fibre	N-free Extract	Ash	Na	K	Na : K Ratio	Ca	Mg	Cl	S	P	Fe	Cu mg.	Mn mg.	Alkali-Alkalinität	Erdalkali-Alkalinität	Total Alkalinität
																		milli. equiv. per 100 g. D.M.		
Danckwortt and Hotzel	Normal hay	—	3·1	—	—	4·62	—	—	—	0·49	0·18	0·90	0·26	0·18	0·04	0·51	—			
	"Lecksucht" hay in Schleswig	—	3·0	—	—	4·59	—	—	—	0·29	0·20	0·84	0·19	0·21	0·05	0·50	—			
Traulsen	Normal hay	10·96	—	—	—	7·83	0·87	2·02	1 : 2	0·69	0·14	1·53	0·14	0·31	—	0·33	23·0	+ 37·76	+ 16·45	+ 54·21
	"Lecksucht" hay in Schleswig	10·87	—	—	—	6·70	0·67	1·28	1 : 2	0·66	0·21	1·15	0·16	0·22	—	0·44	49·0	+ 20·06	+ 27·99	+ 48·05
	Schwarzwald hay, normal	9·51	—	—	—	6·54	0·84	1·60	1 : 2	0·65	0·21	0·39	0·12	0·19	—	0·77	0·0	+ 59·76	+ 32·72	+ 92·48
	"Lecksucht" hay from the Schwarzwald	9·59	—	—	—	7·36	0·60	2·49	1 : 4	0·67	0·18	0·48	0·12	0·23	—	0·37	0·0	+ 68·94	+ 26·41	+ 95·35
Theel	Normal hay :																			
	Average	9·75	2·61	33·72	46·58	7·42	0·17	2·66	1 : 16	0·73	0·24	0·78	0·25	0·25	—	—	—	+ 37·83	+ 32·01	+ 69·84
	Maxmum	13·67	4·03	42·23	50·95	10·70	0·39	4·88	—	1·23	0·34	1·98	0·40	0·39	—	—	—			
	Minimum	7·99	1·71	27·29	40·31	3·56	0·05	0·92	—	0·38	0·11	0·29	0·13	0·14	—	—	—			
Krzywanek and Brüggemann	Normal hay	11·88	2·92	44·91	35·08	5·93	0·15	1·53	1 : 10	0·67	0·28	1·13	0·24	0·24	0·009	—	24·8	− 1·34	+ 33·32	+ 31·98
	"Lecksucht" hay from Schleswig																			
	I	8·31	3·21	29·06	53·62	5·80	0·16	1·81	1 : 11	0·56	0·29	0·91	0·18	0·19	0·011	0·66	27·7	+ 16·48	+ 37·69	+ 54·17
	II	7·85	2·88	29·14	54·05	6·08	0·19	1·67	1 : 9	0·46	0·29	1·30	0·20	0·19	0·019	0·25	24·0	+ 1·93	+ 33·08	+ 35·01
	III	7·81	2·06	30·25	53·09	6·79	0·16	1·65	1 : 10	0·49	0·35	1·24	0·28	0·27	0·021	0·42	24·5	− 3·11	+ 22·27	+ 19·16
	IV	9·78	1·85	32·97	47·60	7·80	0·24	2·11	1 : 9	0·47	0·14	1·55	0·18	0·22	0·020	0·70	9·5	+ 6·75	+ 13·86	+ 20·61
	"Lecksucht" hay Average	8·45	2·49	30·35	52·09	6·62	0·19	1·79	1 : 9	0·50	0·27	1·25	0·21	0·22	0·018	0·51	21·4	+ 5·51	+ 26·73	+ 32·24

Note—The values for the magnesium content of hays II and III have been corrected to conform to an erratum published by the authors in 1942.

Grünigen (1935) observed that lecksucht in grazing cattle in Switzerland was associated with pasture of high K_2O, 6 to 7 per cent. D.M. or more, and low sodium content.

Traulsen (1937) reported that hay from a lecksucht area in Schleswig-Holstein had a low content of potassium and sodium but in the Schwarzwald, where the trouble also occurred, the "affected" hay had a higher potassium and lower sodium and copper content than hay from healthy areas (Table II). Traulsen concluded that low sodium and potassium might be related to the incidence of the disease in Schleswig-Holstein, but Nicolaisen and Seelback (1938) pointed out (p. 56) that the possibility of copper deficiency was not excluded by Traulsen's results.

According to Ibele (1916), quoted by Hagemann and Ohl (1927), "lecksucht" hay in Bavaria is distinguished from healthy hay by its low content of potassium or by the low content of potassium which is available after the chlorine radicle is accounted for. This is reflected in the low alkali-alkalescence of "lecksucht" hay, not greater than 5 to 10 milligram equivalents per 100 g. D.M. and often negative, compared with about 30 m. eq. for good hay (Hagemann and Ohl, 1927). Slagsvold (1933) has more recently emphasised the importance of the low alkali, especially potassium, content of pica-producing hay in Norway (Table I).

Krzywanek and Brüggemann (1939) summarised some published analyses of Schleswig-Holstein and Schwarzwald "lecksucht" and normal hays and were unable to find any abnormality to which the pica could be attributed. These results are shown in Table II.

Lecksucht has been attributed to the frequent use of manures high in potash in Germany and Switzerland (Ostertag and Zuntz, 1907; Truninger and Grünigen, 1935; Grünigen, 1935) but Traulsen (1937) reports that in Schleswig-Holstein lecksucht is controlled by fertilising with potash. Slagsvold (1933) who, on the basis of feeding experiments and hay analyses, attributes the pica which occurs in Norway to deficiency of alkali, expresses surprise that alkali deficiency should occur in an area where there is plenty of seaweed for manuring and where potash is used extensively. He suggests that the explanation may be found in the work of Hedlund (1925) who has shown that on certain sandy soils, but not on clay soils, plants do not take up the expected amount of potassium although the soil may be adequately supplied with this mineral.

The effect of phosphate fertilising has been investigated in Sweden where licking disease is associated with aphosphorosis. Svanberg (1936) reports that in many areas lecksucht responded to fertilising with superphosphate, but at the same time there was propaganda for early cutting and the use of molasses where succulents were not available.

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CHAPTER V

DEFICIENCY OF IODINE

GOITRE

Discussion

Goitre in man and animals occurs throughout the world on a variety of geological formations, but probably most commonly on formations rich in calcium or magnesium. The degree of enlargement of the thyroid is, in general, inversely related to its iodine content, and the disease can usually be prevented and to some extent controlled by administration of iodine.

Clinical studies of human goitre and experimental work with laboratory animals indicate that many factors in addition to a low intake of iodine may co-operate to cause goitre, but it is impossible to judge to what extent, if at all, these factors are concerned in the etiology of goitre in grazing livestock.

There is some evidence that the iodine content of ruminant thyroids shows a seasonal drop in spring, and it has been suggested that this may correspond to the relatively low iodine content of spring pasture observed by some workers. Attempts to correlate the incidence of goitre in livestock with the iodine content of the pasture and hay eaten have, however, not been entirely successful. Although in each area investigated the average iodine content of samples from goitre areas was less than from healthy areas, values deemed healthy in some districts are sometimes lower than those considered goitre-producing in others.

It has been suggested that some external factor, such as deficiency or excess of another mineral, may make a critical level of iodine intake too low. The problem is further complicated by the question of soil contamination. It is doubtful if the results of pasture analysis reflect the iodine intake of the grazing animal since, on the one hand, it is difficult to get samples free from soil and, on the other hand, the animal may be able to utilise some of the iodine contained in the soil eaten along with the pasture. In view of these difficulties, the iodine content of the thyroid is the most reliable index of the need for iodine administration to livestock. No benefit is to be expected if there is no deficiency of iodine to be corrected, and there is evidence that in such circumstances, and especially if there is a deficiency of phosphorus, protein and carotene, continuous administration of doses of the order of 0·05 g. potassium iodide daily to sheep may be harmful. Indiscriminate use of iodised salt licks should therefore be discouraged.

Occurrence and Description of the Disease

Goitre, or abnormal swelling of the thyroid gland, occurs in livestock in the U.S.A., in the states North Dakota, Montana, Oregon, Idaho, Iowa, California, Michigan, Washington, and occasionally in dairy calves in Nebraska and Nevada, and in lambs in Wyoming. "Iodine deficiency" is also reported from Colorado, Minnesota and Wisconsin (U.S. Dept. Agric., 1939). The incidence of goitre outside of the U.S.A. has not been mapped out, but investigations of this disease in livestock have been reported from S. America, Canada, New Zealand, England, Finland, Switzerland, Norway, Sweden and Germany.

The condition occurs in cattle, sheep, goats, horses and pigs, and in areas where it is endemic young animals may be born weak and sometimes hairless.

Experimental Studies

Feeding experiments.—The disease has been controlled by feeding iodine to livestock in affected areas, and administration of iodine to pregnant animals prevents the occurrence of goitre in the young (*Montana Stat. Rep.* 1925 ; Welch and Jungherr, 1927 ; Orr and Leitch, 1929 ; Orr, 1929 ; Kohlschmidt, 1931 ; Bell, 1931 ; Mills, 1934 ; Rodenwold and Simms, 1935 ; von Wendt, 1935 ; Funke, 1935).

Although in most cases goitre in livestock responds to, and can be prevented by, administration of iodine, a condition, characterised by nervous symptoms and enlarged thyroids, occurs in pregnant ewes in S. Africa in which iodine administration has a harmful rather than a beneficial effect. This condition, which occurs on poor, dry, mature pastures low in carotene, phosphorus and protein, is not attributed to deficiency of iodine (Williams *et al.*, 1938 ; Malan *et al.*, 1940). Svanberg (1941) also reports that goitre in calves in Sweden is not prevented by feeding iodine to the cows, but phosphate manuring of the pasture and improving the ration of the cows are beneficial.

Iodine content of the thyroid.—The degree of enlargement of the thyroid is in general inversely related to its iodine content (Orr and Leitch, 1929) ; Hopkirk *et al.*, 1930 ; Sykes, 1934). Mason (1933), in a study of sheep and lamb thyroids, found that this inverse relationship was not clearly defined until the percentage of iodine in the thyroid fell below 0·03 per cent. of fresh weight, the " critical " level of Marine and Lenhart. Hopkirk *et al.* (1930), investigating an outbreak of goitre in lambs in New Zealand, found that thyroids from newborn lambs with enlarged glands had an iodine content of about 0·001 per cent. by weight of gland compared with 0·03 per cent. for a lamb with a thyroid of almost normal size. Piazza (1937), investigating endemic goitre in sheep in Argentina, found that the iodine content of the thyroids of normal lambs in the goitre region varied from 0·17 to 0·25 per cent. D.M., and in the diseased lambs from 0·11 to 0·21 per cent. D.M. Values given by Marine and Lenhart in 1909 for the iodine content of normal and hyperplastic thyroids of ruminants [quoted by Orr and Leitch (1929)] are shown in Table I.

TABLE I

The Iodine Content of Ruminant Thyroids in U.S.A.

	Thyroid	I g. per 100 g.	
		Fresh Weight	Dry Weight
Cattle	Normal . ..	0·112	0·346
	Hyperplastic .	0·004	0·019
Sheep	Normal . .	0·069	0·247
	Hyperplastic .	0·0001	0·0006

Seidell and Fenger (1913) and Martin (1912, 1913) (see Orr and Leitch, 1929) found that thyroids of domestic animals showed a seasonal variation in weight with a maximum in spring which coincided with a minimum iodine content. Orr and Leitch (1929), in reviewing their results, suggest that these observations may be correlated with the relatively low iodine content of spring pasture as observed by Hercus and Roberts. A seasonal variation in the iodine content of thyroid with a maximum in winter was observed in Australian sheep by Dawbarn and Farr (1932) and Duff (1933) in an investigation of thyroids of cattle in Argentina found lower values for iodine in summer than in winter. Mason and Waters (1936) found a maximum iodine content of New Zealand lamb thyroids in January which did not correspond to the Australian results for sheep. They were of the opinion that in lambs the seasonal variation is completely overshadowed by variation due to the rapid growth of the young animal and changes in its iodine requirements. Dawbarn and Farr (1932) found in glands collected from sheep under drought conditions approximately double the percentage of iodine found in glands from the same localities during a good season. Mason and Waters (1936) also found marked differences in the iodine content of glands between the 2 years in which samples were collected, but were unable to correlate the iodine values with the rainfall.

The iodine content of the thyroid can be increased by administration of iodine (Orr and Leitch, 1929 ; Hopkirk *et al.*, 1930 ; Weiser and Zaitschek, 1931 ; Sykes, 1934 ; McClendon, 1939). Mason (1933) failed, except in 1 or 2 cases, to increase the thyroid iodine in lambs by feeding iodised salt licks, but admitted that there was no evidence that the iodine had actually been consumed. There were also indications that iodine had been lost from the licks by decomposition. Dawbarn and Farr (1932) were unable to increase the iodine content of sheep thyroids in Australia by giving iodine licks, but considered that in this case there was probably no initial deficiency of iodine. However, the stability of the licks may also be in question here.

Iodine content of milk.—The iodine content of cow's milk is affected by the intake of iodine ; is sometimes lower in goitre regions than in goitre-free areas ; shows a seasonal variation which has been attributed to the seasonal variation in the iodine content of the pasture or to a seasonal variation in the amount of soil iodine ingested by the grazing animal ; and can be raised by feeding iodine supplements or by the application of iodine-containing fertilisers to the soil (Orr and Leitch, 1929 ; Hercus *et al.*, 1927, 1931 ; Roemmele and Meyer, 1932 ; Shore and Andrew, 1934 ; M.R.C. Rep., 1936 ; McClendon, 1939 ; Matthews *et al.*, 1939 ; Meyer *et al.*, 1940).

Composition of the Soil and Pasture

Soil.—Little appears to have been published from which a correlation between the iodine content of the soil and the incidence of animal goitre could be made, but Hercus *et al.* (1925) state that an inverse relationship does exist. A summary of their soil analyses is given in Table II.

TABLE II

IODINE IN NEW ZEALAND SOILS

District	No. of Samples	Mean I Content μg. per 100 g.
Auckland . .	104	530
Wellington . .	77	480
Canterbury . .	71	90
Otago . . .	219	130
	Total 471	270

These authors have stated that goitre occurs in many species of animals in Canterbury. It is also known to occur in Otago (Mason, 1933). Hopkirk *et al.* (1930), investigating an outbreak of goitre in lambs at Wanaka, New Zealand, found the comparatively low values for soil iodine of 20 to 150 μg. per 100 g. Goitre in livestock is found over widely scattered areas in Oregon (Hartman, 1939) and Powers (1939) has stated that Oregon soils are frequently low in iodine, varying from 50 to 1500 μg. per 100 g.

Hercus *et al.* (1925) observed that a low iodine content of soil was associated with river beds and with alluvial flats where leaching of the soil takes place. Iodine was, on the other hand, relatively abundant in soils overlying igneous rocks. They pointed out, however, that definite correlations between the amount of iodine in the soil and its geological origin were not possible because of the many factors affecting retention of iodine by the soil. Smolik (1935) found no relation between the iodine content of Czechoslovakian soils and the mother rocks from which they originated. Findings differ on the relation between soil acidity and iodine content (Orr and Leitch, 1929; Itano and Tsuji, 1934; Scharrer, 1935), but there appears to be general agreement that soils rich in humus are rich in iodine, that the more colloidal the soil the greater its capacity for holding iodine, and that sandy soils are very poor in iodine compared with clay soils (Hercus *et al.*, 1927; Orr and Leitch, 1929; McClendon, 1939). Mason and Waters (1936), investigating the iodine content of sheep thyroids in Canterbury, New Zealand, found that when the results were grouped according to the geological character of the country the average for basic igneous country was higher than for Miocene and Trias-Jura, and these two in turn gave higher values than Recent alluvium. They were unable to demonstrate a correlation between the iodine content of the glands and the type of soil. The iodine content of the soil can be increased by topdressing with fertilisers containing iodine; loam has marked retentive powers for soluble iodides, in clay the retention is less and in sand it is negligible (Hercus *et al.*, 1927).

Pasture.—In much of the work on the iodine content of pasture attempts to correlate the results with the incidence of goitre have been confined to human goitre. There appears to be no relationship, however, between the incidence of human goitre and the iodine content of pasture (Orr and Leitch, 1929; Orr, 1931; M.R.C. Rep., 1936).

Hopkirk *et al.* (1930), investigating an outbreak of goitre in lambs at Wanaka, New Zealand, found the pasture to be low in iodine, 11 to 17 μg. iodine per 100 g. D.M. compared with 95 μg. for pasture from Islington (Canterbury) sampled at the same time. General analyses showed the Wanaka pasture to be low in chlorine and sodium and high in calcium. It is suggested that at Wanaka some external factor in the shape of excess or deficiency of another mineral may make a critical iodine balance definitely negative. Analyses of sheep thyroids by Orr (1931) showed that the lowest levels of iodine in the thyroid were correlated with low levels of iodine in pasture and milk, but there was no general correlation.

Some attempts have been made to correlate iodine in hay with the incidence of goitre in livestock. Roemmele and Meyer (1932) found that hay from a German farm with a herd of goitrous dairy cattle contained 4·1 μg. iodine per 100 g. compared with 6·2 μg. per 100 g. for hay from a goitre-free farm. Funke (1935), in a study of goitre in young goats on the Glatzer plateau in Silesia, showed that hay from the highlands contained 5·9 μg. iodine per 100 g. dry weight compared with 16·6 μg. for hay from the lowlands. The calcium contents of the hays were similar. Hay from an area of Oregon (U.S.A.), where slight goitre in farm animals is observed, contained 7·5 to 10·0 μg. iodine per 100 g., while alfalfa hay from a severely affected area contained 4·5 μg. per 100 g. (Hartman, 1939).

Orr and Leitch (1929) estimated from analyses of Scottish pastures that the daily intake of iodine for a 110 lb. sheep probably ranged from 120 to 2000 μg., and for a 1100 lb. cow from 3000 to 30,000 μg., but were unable to say whether the lower levels were adequate or not. More recently Mitchell and

McClure (1937) estimated minimal requirements, calculated on the basis of 20–40 μg. iodine per 1000 Calories of the ration, at 50–100 μg. for a 110 lb. sheep and 400–800 μg. for a 1000 lb. cow producing 40 lb. milk daily.

Hercus and Roberts (1927) studied the seasonal variation in the iodine content of New Zealand pasture. They found a maximum iodine content in winter corresponding to the period of minimum growth and a minimum iodine content in spring. Simpson (1930) found the iodine content of New Zealand pastures to be highest in autumn and winter. Orr and Leitch (1929) report that in analyses of samples of Scottish pastures collected in different months during the grazing season an increase in the iodine content was observed in the autumn. Thus, for example, in a series from the same field the figures for August, September and October were respectively, 35, 47 and 58 μg. per 100 g. D.M. It is suggested that iodine accumulates during the period of active growth of the plant and is retained in the mature tissues. On the other hand Fraps and Fudge (1940) have shown that samples of young Bluestem grass grown on various types of soil consistently contained more iodine than samples of mature Bluestem grown on the same types of soil.

The iodine content of pasture also depends on its botanical composition. Differences in the iodine content of pasture species are seen in the analyses of samples from Jealott's Hill farm, where clover and cocksfoot and meadow fescue contained 19 μg. iodine per 100 g. compared with 27 μg. for meadow foxtail and 39 μg. for yorkshire fog (M.R.C. Rep., 1936). Remington and Supplee (1934) analysed samples of fodders grown in S. California and found that legume pasture and hay was the largest contributor of iodine in the cow's diet in this area. The iodine content of legume hays ranged from 32·0 μg. per 100 g. D.M. for sweet clover to 52·6 for soya bean compared with 26·3 μg. for mixed meadow hay. Fraps and Fudge (1940) found the iodine content of young Bermuda grass on some soils was less than that of young Bluestem grass grown on the same soil, and on other soils was greater than that of the young Bluestem grass.

Hercus and Roberts (1927) gave data for iodine in grass and in the soil upon which it was grown in New Zealand which they considered established a correlation between the two. Later, Leitch and Orr (1929) pointed out that the nature of the soil affects the amount of iodine which is available to the plant, and in many cases the total amount of iodine in the soil affords no indication of the amount of available iodine. Examples from Derbyshire are given in which no correlation was found. These two sets of results with the ratio of iodine in soil to iodine in pasture calculated from them are presented in Table III.

TABLE III

IODINE IN PASTURE, RELATION TO IODINE IN SOIL

New Zealand			Derbyshire		
Soil I μg. per 100 g.	Pasture I μg. per 100. g. Fresh Material	Soil I : Pasture I	Soil I μg. per 100 g.	Pasture I μg. per 100 g. D.M.	Soil I : Pasture I
2640	7·2	366·7	343	79	4·3
2000	4·8	416·7	358	84	4·3
1300	7·7	168·8	480	36	13·3
360	3·2	112·5	808	30	26·9
350	2·4	145·8	493	21	23·5
			373	25	14·9
			798	35	22·8
			523	34	15·4

Hopkirk *et al.* (1930) observed little or no correlation between the iodine content of the soil and of the pasture grown on it. In general, the iodine in acid soils appeared to be more readily available to plants than that in alkaline soils. Calculation of the ratios of iodine in soil to iodine in D.M. of pasture from data given by Hopkirk *et al.* for an area at Wanaka where goitre occurred in lambs gives ratios ranging from 1·4 to 11·5. In a later experiment Hercus *et al.* (1931) found no close correlation between the iodine content of soil and pasture, and agreed that the availability of soil iodine must vary from plot to plot.

The experiments reviewed by Orr and Leitch (1929) and McClendon (1939) show that the iodine content of plants can be increased by application of iodine to the soil. Many fertilisers have a relatively high iodine content, and it has been shown that crops from soils treated with manure containing iodine have a higher iodine content than controls from unmanured plots (Hercus *et al.*, 1927). Fertilisers relatively rich in iodine are Chile nitrate, seaweed and guano (Hercus *et al.*, 1925, 1927). Since the effect of these fertilisers does not seem to be commensurate with their iodine content Hercus *et al.* (1927) suggested that the manure stimulates the absorption of iodine by the plant from the soil as well as providing additional easily accessible iodine. As a result of experiments on the effect of potassium iodide and various commercial fertilisers on the iodine content of grass, Hercus *et al.* (1931) obtained results which indicated that the manures increase the iodine content of grass by rendering available the iodine in the soil rather than by virtue of their own iodine. Of the fertilisers tested, superphosphate was most effective. The results are presented in Table IV.

TABLE IV

IODINE IN PASTURE, EFFECT OF FERTILISERS

Plot	Manure Applied to Plot Prior to Sowing with Grass, 15/10/29	Total I in Manure g.	Soil I µg. per 100 g. 5/12/29	Pasture I µg. per 100 g.	
				5/12/29	24/4/30
1	Potassium iodide	0·000765	510	15	21
2	,, ,,	0·038250	670	16	22
3	,, ,,	0·076500	890	26	21
4	,, ,,	0·153000	930	27	33
5	,, ,,	0·306000	1100	34	25
6	,, ,,	0·688500	1310	—	39
7	20 gm. nitrate of soda	0·000474	510	15	20
8	,, Seychelles guano	0·000190	420	13	19
9	,, Nauru phosphate	0·000058	380	20	36
10	,, Superphosphate	0·000304	390	17	28
11	,, special grass manure	0·000152	400	14	—
12	,, blood and bone	0·000002	390	14	21

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N.B.—Orr and Leitch's report (1929), and McClendon's book (1939), review the literature and are not original contributions, but reference is frequently made to these two reviews to avoid recapitulating the original sources.

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CHAPTER VI

DEFICIENCY OF PHOSPHORUS OR CALCIUM

For the past 20 years it has been well-known that in many parts of the world lameness, loss of normal appetite, a craving for bones (osteophagia), osteoporosis and osteomalacia or rickets in grazing cattle associated with impaired reproduction and low milk yield in cows, and stunted growth in young animals, can be prevented by feeding bonemeal or other supplements containing phosphorus. The condition has been recognised as due to a deficiency of phosphorus in the pasture for the following reasons :

1. Affected animals show low blood inorganic phosphorus levels.
2. The condition can be controlled and the occurrence of symptoms, including hypophosphataemia, prevented by feeding supplements of inorganic phosphorus.
3. The condition can be produced experimentally on diets low in phosphorus but otherwise adequate.
4. The " affected " pastures are low in phosphorus.
5. Drought, which reduces the phosphorus content of the pasture, is associated with a high incidence of the disease.
6. Increasing the phosphorus content of the pasture by application of phosphatic fertilisers has a beneficial effect on cattle in areas where the pasture is low in phosphorus.

Sheep are not so susceptible to phosphorus deficiency as cattle, and this has been attributed to their less massive skeleton and to their habit of selectively grazing the meristematic tissues in which much of the phosphorus of the plant is stored. True osteomalacia has not been reported in grazing sheep, but clinical signs of aphosphorosis have been observed.* Horses also are not very susceptible to aphosphorosis, but in areas in S. Africa where cattle are affected horses may be of poor stature, and in America, cases of lameness and hypophosphataemia in horses which responded to treatment with bonemeal have been reported under drought conditions. In areas where pasture is low in phosphorus the protein content is also low, and is very often the factor limiting the growth and productivity of both cattle and sheep. It has been suggested that the resulting low rate of growth due to protein, and other deficiencies, may sometimes prevent the appearance of symptoms characteristic of aphosphorosis in cattle grazing on pasture which does not appear to contain enough phosphorus for normal requirements. This would appear also to be an additional reason for the rarity of clinical aphosphorosis in sheep on pastures low in phosphorus.

The botulism which occurs in cattle in phosphorus deficient areas has been shown to be due indirectly to phosphorus deficiency, since the osteophagia sometimes leads to ingestion of carrion infected with *Clostridium botulinum*, but in the case of botulism in sheep in the phosphorus deficient areas of West Australia it has been shown that a protein deficiency, sarcophagia, is responsible for the ingestion of infected carrion.

Probably another complicating factor in phosphorus deficient areas is the tendency towards low carotene content in pasture under conditions of drought which leads to symptoms of vitamin A deficiency in grazing cattle.

Theiler and his associates in South Africa, to whose credit lies most of the work on phosphorus deficiency, have shown that the production of rickets in cattle depends only on deficiency of phosphorus, although the ratio of phosphorus to calcium may play a part in the production of the

* In Canterbury, New Zealand, true rickets has occurred in sheep associated with consumption of green oats. Intake of calcium and phosphorus was adequate and lack of vitamin D was suspected (Fitch, W. N. 1943, *Aust. Vet. J.*, **19**, 2).

associated clinical symptoms. Some European workers, on the other hand, have consistently emphasised the importance of the *Erdalkali-Alkalinität* ($CaO + MgO - P_2O_5$) in the etiology of bovine osteomalacia and rickets, and have produced these conditions experimentally on diets adequate in phosphorus but with an adverse ratio of calcium and magnesium to phosphorus. It has been pointed out, however, that the difference between the findings of these two schools of thought probably lies in the fact that under the South African conditions, both natural and experimental, bright sunlight provided an abundance of vitamin D, whereas in the European experimental work, the animals were stall fed and there was an insufficiency of vitamin D.

Pastures with a very low content of calcium have been reported, but there is no evidence that grazing animals suffer from a straight acalcicosis. Attempts to produce calcium deficiency experimentally indicate that cattle and sheep are comparatively immune. But ill health and bone deformity occur in sheep on a low calcium intake associated with lack of sunshine or vitamin D, for instance the bent-leg of Scotland. Horses fed roughage low in calcium along with concentrates such as bran and cereals high in phosphorus develop a bone disease known as osteodystrophia fibrosa. A similar condition has been produced experimentally in calves fed a ration low in calcium with a low calcium to phosphorus ratio, and inefficient milk production and brittleness in bones of dairy cows in Florida have been traced to the same cause. These conditions have all been cured or prevented by administration of calcium and appear to be due to reduction of the utilisation of an already low calcium intake by lack of vitamin D in the case of the sheep and the large excess of phosphorus in the other cases.

A detailed account of the investigations of calcium and phosphorus deficiency is not included here because recent literature is so extensive and yet tends only to confirm the earlier findings which have already been adequately reviewed. A list of useful reviews is appended.

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CHAPTER VII

HYPOMAGNESAEMIC TETANY

GRASS TETANY, GRASS STAGGERS OR LACTATION TETANY

Discussion

A form of tetany in livestock characterised by low blood magnesium has been reported in many parts of the world. It is commonly called either lactation tetany, because it occurs most often in lactating cows, or grass tetany or grass staggers because of its frequent association with turning out to pasture in spring. These are misnomers, however, because the disease also occurs, although less frequently, in dry cows, bullocks, calves and breeding ewes, and because it also appears in stall fed cattle during winter.

In general, clinical symptoms of grass tetany are associated with low values for serum magnesium, but high values have been demonstrated in some affected animals and low values have been observed in unaffected cows at the time of occurrence of clinical cases of tetany. These anomalies may be explained on the basis of Tufts and Greenberg's (1937–38) work on rats.

These workers found that rats on a diet deficient in magnesium showed a sharp reduction in serum magnesium, followed, after an interval the length of which varied directly with the magnesium content of the diet, by a rise to normal levels. Thereafter the level gradually declined but did not again reach the previous minimum. Symptoms of magnesium deficiency did not appear when serum magnesium was minimal but during the second decline. In the rats studied the minimum was reached on the third day, which was shown by Duckworth and Godden (1941) to coincide with exhaustion of the mobilisable fraction of the magnesium of the skeleton. It is therefore possible that cows showing symptoms of tetany will have passed the minimal levels of serum magnesium before examination and may be at any stage in the second phase with normal or falling values, and that cows, apparently unaffected, showing low values for serum magnesium may not reach the second phase with clinical symptoms during the period of observation or at all. On the other hand, there may be failure of mobilisation of skeletal magnesium as discussed below.

Low serum calcium is often associated with low blood magnesium in cases of grass tetany, and injection of calcium and magnesium salts together has proved beneficial where the disease had not progressed too far before the treatment was begun.

There is no evidence that hypomagnesaemic tetany results from a deficiency of magnesium in the diet of affected animals. Pasture analyses indicate a sufficiency of magnesium, but in some areas the manganese content of the pasture is high and erroneous conclusions may have been reached concerning the adequacy of the pasture magnesium since Duckworth and Godden (1938) have shown that, with the methods of analysis used, co-precipitation of manganese and magnesium would give a fictitiously high value for magnesium. In other areas where it has definitely been established that the manganese content of the pasture is not abnormally high this argument would not, of course, apply. Too much emphasis should not be laid on dietary adequacy, however, for, as Duckworth (1939) pointed out, calves become hypomagnesaemic and exhibit classical symptoms of magnesium deficiency on a diet composed exclusively of milk. Replacement of part of the milk by cereals yielding about the same magnesium intake could cure or prevent the condition.

Green (1939) has suggested that the disease may ultimately prove to be an endocrine disturbance. To this suggestion the more recent studies of Duckworth (1939), Duckworth *et al.* (1940), Duckworth and Godden (1941), Duckworth and Warnock (1942–43) and Duckworth and Godden (1944) give some support. The magnesium content of the rat skeleton was found to be a highly labile reserve, the outflow of which occurred rapidly in response to a dietary deficiency. Re-establishment of the reserve after depletion was, however, slow, and depended on the rate of mineral deposition, magnesium not being capable of entering the bone salt crystal independently of calcium and phosphorus. Thus, failure of magnesium to be liberated to remedy a plasma deficit might indicate previous depletion without restoration of the normal reserve. The limited data available, however, on the magnesium content of the organs, bones and milk of affected cows give no indication of any abnormality. Duckworth and Godden (1941), discussing the possible involvement of the endocrine system in the liberation of skeletal magnesium, are of the opinion that the level of magnesium in the fluid phase of the bone cannot alone be responsible for removal of the magnesium ion from the bone crystal; hence, disturbance of the mobilising endocrine mechanism may be concerned in the inability of an animal to maintain a "normal" level of serum magnesium.

The cause of grass tetany has also been sought in other pasture abnormalities. Sjollema observed that affected pastures in Holland were high in protein and in potassium nitrate, but was unable to produce clinical symptoms experimentally by feeding diets high in protein or in nitrate although some reductions in the levels of serum calcium and magnesium were obtained. Hopkirk and his co-workers have suggested that the high phosphorus and low calcium and possibly the high protein content of affected pastures may be related to the occurrence of tetany in grazing cows in New Zealand.

It is possible that in different areas different factors may be contributory causes of grass tetany, and although no obvious connection exists between the disturbance in magnesium metabolism and the mineral composition of the pasture, there is not sufficient evidence of any kind to justify the exclusion of grass tetany from this review of diseases attributable to deficiencies or excesses of minerals in the pasture.

Occurrence and Description of the Disease

A form of tetany has been reported in Holland, Schleswig-Holstein, Norway, Britain, Ireland, America, New Zealand and Australia. In New Zealand the soil of the badly affected Waikato district includes a high proportion of air or water-borne pumice compared with other districts, and the land is comparatively light, responding readily to fertilisers (Hopkirk *et al.*, 1933). In England Blakemore *et al.* (1937) found that in general there was little or no connection between the type of soil and the distribution of the disease in affected counties.

The disease occurs mainly in lactating cows, often shortly after calving, but it has also been reported in dry cows, bullocks, calves and breeding ewes.

The initial signs in cows are usually restlessness, nervousness, loss of appetite, and hyperexcitability of the neuromuscular mechanism. Respiratory and pulse rates are increased and convulsions occur during which the animals are in a state of intense excitement. Attacks do not always end fatally, but death may occur within an hour or in two or three days. The symptoms of affected sheep described by Blumer *et al.* (1939) closely resemble those of cows.

The incidence of the disease varies from year to year. In Holland, Britain, Schleswig-Holstein and America the disease usually occurs in milk cows recently turned out to graze on a lush growth of spring grass, although outbreaks have occurred in cows on pasture in the autumn (Sjollema, 1932 [1]; Warringsholz, 1931) and in stall fed cows in winter (Blakemore and Stewart, 1932–33; Nicholson and

Shearer, 1938; Nolan and Hull, 1941). In New Zealand the disease occurs, usually between July and September, in cattle which are pastured throughout the year. The cows are fed supplements of hay, silage and sometimes roots during winter and spring, and, shortly after calving, are transferred to fresh pasture which has not been grazed after being manured in the autumn (Hopkirk *et al.*, 1933). In Ireland the disease has also been reported in winter in cows that are never housed and whose sole diet is pasture (Nicholson and Shearer, 1938).

In Queensland, cases in cows have followed continuous feeding on growing oats and barley or on natural pastures rich in clover. A history is often given that cows were put into a paddock of luscious, quickly growing feed from a fortnight to a month before the first cases occurred (Mulhearn, 1936). Lush grazing conditions in Australia produced by winter rains are associated with a high incidence of grass staggers in breeding ewes (Blumer *et al.*, 1939).

Experimental Studies

Composition of the blood.—Hypomagnesaemia is a constant feature of grass tetany in cows. Sjollema (1930) recorded values for 55 cows ranging from 0·1 to 1·16 mg. magnesium per 100 c.c. serum with an average of 0·5 mg. Similar low values have been obtained by Dryerre (1932), Blakemore and Stewart (1932–33), Hopkirk *et at.* (1933), Allcroft and Green (1934), Nicholson and Shearer (1938) and Nolan and Hull (1941). Blakemore and Stewart (1932–33) observed that the values for magnesium had not returned to normal within 36 days after the passing of acute symptoms.

There appears to be a tendency for low serum magnesium to be associated with low serum calcium in grass tetany. Sjollema (1930) found an average value of 6·65 in the 55 cases studied; Nicholson and Shearer (1938) found values of 5·73 and 7·33 mg. in 2 cases, and Nolan and Hull (1941) reported an average of 6·6 mg. per 100 c.c. serum for 12 affected cows. Blakemore and Stewart (1932–33) found values of 6·40 to 7·33 mg. calcium per 100 c.c. serum for 4 acute or subacute cases, but observed a rapid return to normal after the acute symptoms passed. The work of Dryerre (1932), Allcroft and Green (1934) and Hopkirk *et al.* (1933), however, showed that low values are not always found. Dryerre (1932) reported values varying from 5·6 mg. calcium to 11·0 mg. with an average of 9·3 mg. calcium, and Allcroft and Green (1934) obtained values ranging from 4·7 to 10·8 mg. calcium for 18 affected cows. Sjollema's results, although low on the average, showed values as high as 9·5 mg. calcium but he suspected these might have been due to previous inflation of the udder. Sjollema (1932 [2]) pointed out that the ratio of calcium to magnesium in the serum of affected cows is about 14 compared with 5·6 for normal cows, and Dryerre (1932) also stressed the significance of the high calcium to magnesium ratio in grass tetany. Allcroft and Green (1934), and Nicholson and Shearer (1938) did not, however, find any correlation between the calcium to magnesium ratio and the onset of the disease. Sjollema (1930) found that values for serum phosphorus fluctuated much more in affected than in unaffected cows. In general the calcium to phosphorus ratio in grass tetany was low. Hopkirk *et al.* (1933) found that hypocalcaemia with hyperphosphataemia were sometimes associated with the low blood magnesium of grass tetany. Sjollema (1930) also analysed the blood of affected cows for potassium, sodium and chlorine. The first seemed to be within normal limits but the values for sodium and chlorine appeared to fluctuate rather more than in normal cows.

Because of the characteristic low blood magnesium, investigations into the etiology of the disease have tended to centre round this mineral. Blakemore and Stewart (1934–35) found in districts where grass tetany is prevalent a seasonal drop in serum magnesium in cows put out to pasture in early spring, and the duration of the period of low blood magnesium coincided with the 6 weeks period during which most cases of grass tetany have been observed. They also found that this drop in serum magnesium could be prevented by feeding supplements of magnesium for about a month before the animals went out to pasture. The drop in blood magnesium appeared to be confined to areas where grass tetany occurs, although only one of the experimental cows developed the disease. This cow, although it did not have the lowest serum magnesium, was the only one with a low serum magnesium associated with hypocalcaemia, the respective values being 1·60 and 6·33 mg. per 100 c.c. Allcroft and Green (1938), in a study of 89 Hereford cows kept outdoors on pasture throughout the year and fed supplements of hay and a mineral lick in winter, found a seasonal drop in the magnesium level of the blood which reached a minimum in winter and rose again over the period when fresh growth of grass was becoming apparent. Normal levels were reached in February. Values as low as 0·5 mg. magnesium per 100 c.c. serum were observed in December, but there was no sign of tetany and no evidence of hyperexcitability. Administration of magnesium oxide reduced the December fall but did not altogether prevent it. No change occurred in serum calcium. The authors emphasise that the data show a complete dissociation of hypomagnesaemia from spring flush of grass, turning out to pasture, lactation and any manifestation of tetany. Another group of cows of mixed breed in another paddock of the same farm did not show such a marked reduction in serum magnesium. A study of a group of 25 cows over the

complete year from June 1937 to June 1938 showed that the rise from the December minimum which occurred in January and February was not followed by a secondary spring fall, but in this connection it is pointed out that the incidence of grass tetany was unusually low throughout England as a whole in the spring of 1938. Allcroft and Green do not offer any explanation of the seasonal variation in serum magnesium, but they suggest that when changes in blood calcium and magnesium associated with parturition coincide with a seasonal minimum magnesium level in the serum the onset of lactation tetany may be precipitated.

Blakemore *et al.* (1937) found that pasture in Lincolnshire, where grass tetany commonly occurs, had an unusually high manganese content. They therefore examined the manganese content of the blood of cattle grazing on such pastures and found that 6 cows with an average manganese of 0·0015 mg. per 100 c.c. serum before being turned out to grass showed an average level of 0·1599 mg. after 20 days on the "affected" pasture. The average for 8 cows grazing on an unaffected area was 0·0025 mg. manganese. Experiments in which sheep and cows were fed manganese in the amounts which they would have consumed had they been on such pasture showed that a temporary depression in serum magnesium resulted but not to the extent which is usually associated with lactation tetany. No symptom of ill health was observed. Administration of lethal doses of manganese resulted in an increase of serum magnesium. Green (1939) states that co-operative experiments carried out at Cambridge, Weybridge and Moredun showed that administration of manganese chloride did not specifically lower the blood magnesium in either cattle or sheep, and that no reduction was produced by raising the blood manganese even a hundredfold by direct injection. Oral administration of massive doses of easily available manganese with the food did not alter the manganese content of the blood to any significant extent. At this point consideration should be given to the work of Duckworth and Godden (1938) who showed that administration of manganese chloride by stomach tube to rabbits caused an apparent rise in serum magnesium which they showed was mainly due to co-precipitation of manganese with magnesium in the estimation of magnesium. They pointed out that the apparent increases in serum magnesium obtained by Blakemore in response to lethal doses of manganese were probably fictitious, and due to co-precipitation of manganese and magnesium. It would appear that in the experiments referred to by Green the negative results obtained might be explained on this basis unless manganese was precipitated before the magnesium was estimated.

Composition of tissues.—Cunningham (1936) analysed milk, bones and organs from cows with grass tetany, and found that in all the magnesium was within normal limits. The magnesium content of urine was low, as would be expected with the low blood magnesium, and showed no evidence of a pathological loss of magnesium.

Feeding experiments.—Sjollema found that grass tetany could be cured by injection of calcium and magnesium salts, but pointed out that because of the rapid onset and swift course of the disease it is very often impossible to prevent a fatal issue, even with the most careful treatment (Sjollema 1932,[2] 1932[3]). Broersma (1933) and Kuipers (1933) have also found treatment with calcium and magnesium together curative, and Blumer (1939) reports success with affected sheep in Australia. Hopkirk *et al.* (1933), on the other hand, found that this treatment gave no useful results, but later reported (Duckworth, 1939) that the occurrence of grass tetany could be prevented by feeding silage to which dolomite, a double carbonate of calcium and magnesium, had been added at the time of ensiling.

Seekles and Sjollema (1932, 1933) suspected that the high protein and nitrate contents of spring pasture were involved in the etiology of the disease, but were unable to reproduce grass tetany artificially in cows by feeding high protein and high nitrate diets. Administration of nitrate solution, pasture grass high in protein and nitrate, and earthnut meal all caused reduction of serum calcium and magnesium, and variations in serum inorganic phosphorus, but protein-free extracts of grass and oats, and peptone had similar effects, and soya beans and wheat gluten as sources of protein did not have these effects on the blood constituents. It should be pointed out, however, that negative results in such experiments may be due to mobilisation of the skeletal reserves.

Composition of the Pasture

Holland.—Sjollema (1931 [1, 2]) analysed "affected" pasture and found that it contained a very high percentage of crude protein. Of 372 samples analysed, 10 contained over 30 per cent. crude protein, 68 contained between 25 and 29·9 per cent. and 173 contained between 20 and 24·9 per cent. crude protein on a dry matter basis. The nitrate content was also high, up to 2 per cent. potassium nitrate on a dry matter basis. Some of the pasture samples, all of which were taken immediately after the occurrence of cases of lactation tetany, were analysed for minerals, and a summary of the results reported for calcium, phosphorus and magnesium (Sjollema, 1932 [1]) are presented in Table I.

TABLE I

MINERALS IN "AFFECTED" DUTCH PASTURES (PER CENT. D.M.)

	Spring			Autumn		
	No. of Samples	Range	Average	No. of Samples	Range	Average
CaO	69	0·38–0·95	0·64	5	0·42–0·62	0·50
P_2O_5	67	0·50–1·37	0·96	6	0·92–1·17	1:01
MgO	68	0·12–0·32	0·25	6	0·22–0·33	0·28
$\frac{CaO}{P_2O_5}$			$\frac{1}{1·5}$			$\frac{1}{2}$

The magnesium and phosphorus contents were about normal for cultivated pasture, but the samples were on the whole low in calcium and contained only very small amounts of sodium, mostly about 0·1 per cent. Na_2O. The potassium was very high in most samples, with an average of about 4 per cent. K_2O. Hence, the ratio potassium to sodium was very wide, in some cases greater than 50 : 1. Later Sjollema (1932 [2]) suggested that ill-balanced winter rations, containing an excess of cereals and little hay, were predisposing causes of grass tetany, giving rise to a condition approaching, in some cases, latent tetany. The sudden transition from such rations to spring grass high in protein, nitrate and potassium, and relatively low in calcium and sodium precipitated the onset of the disease. Unsuccessful attempts to produce the condition artifically by administration of high protein and high nitrate diets have been reported above.

Broersma (1932, 1933) and Kuipers (1933) have suggested a possible correlation between excessive use of artificial nitrogenous fertilisers and the incidence of grass tetany in Holland, but this has not been established.

Britain.—Blakemore and Stewart (1934–35) investigated the magnesium content of pasture in districts where lactation tetany occurs. The range on a dry matter basis for 6 disease-free fields was 0·61 to 1·01 per cent. magnesium oxide (average 0·81) compared with 0·65 to 1·03 (average 0·85) for 8 fields where lactation tetany was prevalent. Later Blakemore *et al.* (1937) pointed out that the disease was generally associated with poor types of grass such as yorkshire fog, sweet vernal, yarrow, floating foxtail and fiorin, though the actual amount of grass available was usually high. Chemical analyses of grass samples from affected farms in Lincolnshire, Northamptonshire and Herefordshire showed no abnormality in composition of the organic matter, but the pastures were abnormally high in manganese, 54–132 mg. per 100 g. D.M. compared with 1·6 to 7·8 mg. per 100 g. D.M. of samples from unaffected pastures in these counties and 4·1 to 10·6 mg. in Cambridgeshire. Steps were taken to ensure that the samples were free from soil contamination. It was suggested that the high manganese content of the pasture might be responsible for the high manganese levels in the blood of cows turned out to graze on affected farms and, since feeding sheep and cows with amounts of manganese equal to what they would have consumed on such pasture produced a temporary fall in blood magnesium, it was concluded that the high manganese content of the pasture, in Lincolnshire at least, might be the cause of the low blood magnesium observed in the cows grazing in grass tetany areas. According to Green (1939) this point of view was subsequently abandoned by Blakemore.

Ireland.—Nicholson and Shearer (1938), investigating 4 outbreaks of lactation tetany in Ireland, observed that pasture on farms where outbreaks occurred was composed largely of poor quality grasses, principally yorkshire fog, cocksfoot and foxtails, with an abundance of common sorrel. They thought that this flora indicated lack of available calcium in the soil. Three of the outbreaks occurred in January (*a*) in cows never housed but fed solely on pasture ; (*b*) in cows which during the winter were housed at night, pastured during the day, and given supplements of concentrates and good quality hay ; (*c*) in cows which were continuously housed from the middle of October to the beginning of May and fed concentrates and hay. Analyses of grass and hay from affected farms showed a badly-balanced mineral content, high manganese content, and magnesium within the normal range (Table II). Analyses of the concentrates are not given, but it is stated that the mixture fed did not appear to possess a well-balanced mineral content but was probably rich in phosphorus and low in calcium.

Other outbreaks of the disease are reported in cows in May and June, a few weeks after being turned out to pasture, and from February to April in cows which were stall fed until the beginning of March and then turned out to graze during the day and, towards the end of March, left out and fed good quality hay and concentrates in addition to pasture. Analyses of feeds were not made in these cases.

TABLE II

PROTEIN AND MINERALS IN GRASS AND HAY (PER CENT. D.M.)

Outbreak	Crude Protein	CaO	P_2O_5	MgO	Mn	$\frac{CaO}{P_2O_5}$
(*a*) grass	16·23	0·48	0·83	0·272	0·0813	$\frac{1}{1·7}$
(*b*) grass	14·22	0·48	0·52	0·353	0·0656	$\frac{1}{1·1}$
hay	6·94	0·38	0·29	0·222	0·0568	$\frac{1}{0·8}$
(*c*) hay	7·81	0·67	0·36	0·207	0·0495	$\frac{1}{0·5}$
(*d*) pasture upon which no outbreak occurred but on same farm as outbreak (*a*)	13·66	0·57	1·03	0·283	0·0583	$\frac{1}{1·8}$

New Zealand.—Aston's analyses of pasture samples from areas affected and unaffected with grass staggers are quoted by Hopkirk *et al.* (1933). The calcium, phosphorus, magnesium, manganese, sodium, potassium, and nitrogen values and the CaO : P_2O_5 and potassium to sodium ratios calculated from them are presented in Table III. In the 1931 Waikato samples taken while the trouble was at its height there was a comparatively high ratio of phosphate to lime which suggested the use of the best strains of perennial ryegrass. It is pointed out that grass tetany occurs mainly in those districts of New Zealand which use a good type of ryegrass. The Waikato pastures were high in protein whether "affected" or unaffected, and pasture from an "affected" farm in Wairarapa also showed a high protein content. The potassium to sodium ratio in both "affected" and unaffected pasture was lower than in affected Dutch pastures, and was not considered to have any significance. According

TABLE III

PROTEIN AND MINERALS IN NEW ZEALAND PASTURE (PER CENT. D.M.)

District	Samples	Time of Sampling	Protein (N × 6.25)	CaO	P_2O_5	MgO	Mn	Na_2O	K_2O	$\frac{CaO}{P_2O_5}$	$\frac{Na}{K}$
Unaffected											
Waikato	7 pastures	Sept. 1929 end of eclampsia season	28·1	0·90	0·84	0·39	0·022	0·37	3·51	$\frac{1}{0·93}$	$\frac{1}{10·}$
Taranaki	59 of Ca, P, N. 6 of others	Oct. 1929 good and fair pasture	22·6	0·96	0·74	—	—	0·69	2·69	$\frac{1}{0·77}$	$\frac{1}{4·4}$
Wairarapa	16 mixed	1929	26·5	0·76	0·92	0·46	0·023	0·32	3·45	$\frac{1}{1·21}$	$\frac{1}{11·9}$
Waikato	3 farms	Sept. 1931 during eclampsia season	33·8	0·74	1·11	—	—	—	—	$\frac{1}{1·50}$	—
Affected											
Waikato	9 farms	Sept. 1929	27·9	0·91	0·87	0·39	0·019	0·29	3·76	$\frac{1}{0·96}$	$\frac{1}{14·9}$
Wairarapa	1 farm	Oct. 1929	32·8	0·60	1·09	0·50	0·014	—	4·52	$\frac{1}{1·82}$	—
Waikato	20 farms	Sept. 1931	31·5	0·68	1·22	—	—	—	—	$\frac{1}{1·79}$	—

to Hopkirk *et al.* the abnormality most likely to be associated with grass tetany on New Zealand pasture was the high phosphorus and low calcium with perhaps the high content of protein of an easily assimilable nature as a secondary influence.

Cunningham (1936) investigated the magnesium content of samples of New Zealand pasture obtained periodically throughout the year from 4 farms situated in grass staggers areas. The magnesium oxide in 39 samples ranged from 0·27 to 0·64 per cent. D.M. with an average of 0·40. The average for samples collected from July to September, when grass staggers is most prevalent, and of samples taken during grass staggers outbreaks on 4 other farms was 0·34 per cent. magnesium oxide. This value, although slightly subnormal, was not considered low enough to account for the extremely low blood magnesium characteristic of grass staggers. The magnesium oxide content of feeding-stuffs grown on the farms ranged from 0·24 to 0·35 per cent. D.M.

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CHAPTER VIII

SELENIUM EXCESS

ALKALI DISEASE AND BLIND STAGGERS

Discussion

Probably the first authentic record of selenium poisoning in livestock is that of Madison in 1856 who described a fatal disease in army horses in Nebraska characterised by loss of hair from the mane and tail and soreness of the feet. A similar condition was later described in cattle and sheep to which the name "alkali disease" was given because it was attributed to excessive consumption of alkaline waters. The name has persisted although this theory has long been disproved. A more acute form of the disease was also observed in horses, cattle and sheep to which the name "blind staggers" was applied. In some states, for example in South Dakota, the chronic form predominates, and in other areas, as in Wyoming, the acute form is more common.

In experiments with rats, begun in 1929, Franke showed that grains from affected areas produced symptoms and lesions similar to those occurring naturally in livestock. In 1933 Robinson discovered abnormal amounts of selenium in these toxic grains, and subsequent investigations with rats showed that inorganic selenium had the same effect as toxic grain and precipitation of selenium from the

grain rendered it non-toxic. It was further shown that administration of inorganic selenium to pigs and horses produced symptoms and lesions similar to those seen in alkali disease.

This discovery led to extensive investigations of the selenium in rocks, soils and vegetation. It was found that alkali disease results from consumption of forage plants containing between 4 and 25 p.p.m. of selenium and predominates on cultivated pasture. Blind staggers, which predominates on range pastures, results from consumption of certain range plants belonging to the genera *Astragalus*, *Stanleya*, *Oonopsis* and *Xylorrhiza* which seem to have a high requirement of selenium and may contain several thousand p.p.m. The presence of such plants is used as an indication of seleniferous soils. Livestock usually avoid them except when over-grazing or lack of other vegetation restricts their choice, usually with fatal results.

These seleniferous weeds, in addition to causing blind staggers or acute selenium poisoning, contribute indirectly to the occurrence of alkali disease, or chronic selenium poisoning, because they can take up selenium which appears to be unavailable to ordinary forage plants and convert it into a form which, when returned to the soil by leaching or decay, can be absorbed by forage plants. So far there has been no satisfactory explanation of this behaviour.

Seleniferous vegetation appears to occur almost exclusively on Cretaceous formations and, as far as can be judged from published records, does not constitute a problem in livestock outside the continent of America. These formations usually gave rise to soils high in selenium, but the toxicity of the vegetation grown on them depends on such factors as the chemical form in which the selenium occurs and the amount of rainfall or irrigation.

Attempts to find a dietary factor or factors which would prevent or reduce the toxicity of seleniferous rations have been reviewed by Moxon and Rhian who point out that, in view of the species differences shown in this work with rats and dogs, experimental work with farm animals is needed before any practical application can be recommended. Non-dietary detoxifying factors have also been tried of which arsenic has given the most promising results with laboratory animals. Animals suffering from selenium poisoning have been shown to store selenium in their tissues and Moxon, who has shown that administration of certain aromatic compounds to steers on a seleniferous range caused an increased urinary output of selenium, recommends further investigations along these lines.

Little is known of the mechanism of poisoning by selenium but there is some evidence that it inhibits the activity of certain enzymes.

Occurrence and Description of the Disease

" Alkali disease " has been known for many years as a chronic condition in horses and cattle grazing on the semi-arid Great Plains of the U.S.A., probably the first mention of it being among army horses in Nebraska in 1856 (Maddison, 1860). " Blind staggers," which is believed to be an acute form of the same disease, was reported in Kansas as early as 1891 (Mayo). More recent investigations have shown that these conditions occur in Wyoming, S. Dakota, Colorado, Mexico, Montana and Nevada, as well as in Nebraska and Kansas in varying proportions ; " alkali disease " is more common in S. Dakota and " blind staggers " in Wyoming. In Canada the chronic type has been reported from Saskatchewan, and areas in Alberta and Manitoba have been described as " toxic " or affected. These conditions appear to be confined to soils derived from Cretaceous shales or limestone, particularly the Pierre, Niobrara, Morrison and Greenhorn selenium-bearing formations (Beath *et al.*, 1934 ; Franke *et. al.*, 1934 ; Knight and Beath, 1937 ; Moxon, 1937 ; Williams *et al.*, 1941 ; Byers *et al.*, 1935, 1936, 1938).

" Alkali disease " affects cattle, horses, sheep and pigs, and is characterised by dullness and lack of vitality ; emaciation ; depraved appetite ; rough coat in horses and cattle ; loss of hair from the mane and tail of horses and from the switch of cattle ; soreness and sloughing of the hoofs and stiffness of the joints. Atrophy of the heart, atrophy and cirrhosis of the liver, and erosion of the long bones also occur. There is a high incidence of anaemia, and according to Moxon and Rhian (1943) the haemoglobin content of the blood can be used to detect the earlier stages of " alkali disease " in cattle. In severe cases the haemoglobin may drop as low as 7 g. per 100 c.c. The sucking young are often affected. There is evidence of interference with the circulation of the blood resulting in freezing of the feet and lower parts of the legs and tail when affected animals are exposed to extreme cold, a condition referred to as " frozen feet " by Saskatchewan ranchers.

" Blind staggers " may develop in cattle in a comparatively short time. The affected animal has a tendency to wander from the herd ; its vision seems to be affected and to become progressively worse. There is depraved appetite for bones and metallic objects. The final stage is characterised by almost complete blindness and varying degrees of paralysis. The animal salivates freely, grates its teeth and grunts. Death is due to respiratory failure. Eye involvement is not observed in all acute cases ;

the usual changes are swelling of the eyelids and profuse lachrymation ; there may be a slight turbidity of the aqueous humour or the entire cornea may become white and opaque, sometimes with haemorrhagic areas. A condition somewhat similar has been observed in sheep. There is a high incidence of anaemia in " blind staggers " as well as in " alkali disease."

Experimental Studies

Feeding experiments.—" Alkali disease," as the name suggests, was at first attributed to the drinking of alkaline waters, but experimental work did not support this view (Larsen *et al.*, 1912 ; Larsen and Bailey, 1913 ; Beath *et al.*, 1934). Later the fodder became suspect, and in a series of investigations begun in Dakota in 1928 it was shown that grain from affected areas was toxic to rats. The rats responded immediately by restricting their food consumption. In the first animals to die the gross pathology was not clear in comparison with cases dying later and termed chronic. The main findings in the chronic cases were atrophy, necrosis and haemorrhagic changes in the liver. These animals also developed a progressive anaemia, their haemoglobin reaching very low levels before death. The anaemia was not due to inanition since control rats on similar intakes of non-toxic grain did not become anaemic. The animals often showed bone conditions said to resemble vitamin D deficiency although the diets were adequate in that respect and the controls had developed normally. The acute cases showed high haemoglobin values attributed to anhydraemia (Franke, 1934 ; Franke and Potter, 1934). Samples of the toxic wheat used were shown by Robinson (1933) to contain selenium. Later, Franke (1934 [2]) showed that the toxicant was mostly in the protein fraction of the grain, and this was later confirmed by Horn *et al.* (1936).

The main symptoms of poisoning in rats caused by feeding toxic grains have been reproduced by feeding inorganic selenium (Franke and Potter, 1935 ; Munsell *et al.*, 1936 ; Martin, 1936 ; Schneider, 1936 ; Franke and Moxon, 1937 ; Franke and Painter, 1938 ; Gortner and Lewis, 1939). Munsell *et al.* (1936) showed that the inclusion of wheat containing selenium in the diet of rats has a detrimental effect on growth and reproduction in direct proportion to the amount of selenium supplied. Selenium added to the diet in the form of selenious acid was toxic in proportion to its concentration in the diet. Franke and Potter (1936) also demonstrated the adverse effect on growth and reproduction in rats of toxic grain, and showed that young rats were more susceptible to the toxicant than older rats. Precipitation of selenium by means of mercuric chloride renders the toxic protein hydrolysate from toxic grain innocuous to rats, which indicates that either selenium is the sole toxicant or other toxicants, if present, are likewise precipitated by mercuric chloride. No toxicant was removed from either toxic proteins or their hydrolysates by organic solvents so that nearly all plant toxicants known at the time could be eliminated (Franke and Painter, 1935).

The presence, in addition to selenium, of other elements such as vanadium and molybdenum has been reported in toxic vegetation (Byers, 1934 ; Beath *et al.*, 1935, 1939). Beath *et al.* (1935), reporting the occurrence of molybdenum in Wyoming vegetation, suggested that any effect of this molybdenum on livestock was probably obscured by the selenium which occurred in most of the mineral-bearing plants. They therefore carried out 2 tests to gain more specific data on the effect of molybdenum. A partial crop of barley stems, leaves and immature heads was secured from an acre of land the surface soil of which had been mixed with 75 lb. sodium molybdate before sowing. Composite samples gave 89 p.p.m. of molybdenum, and this barley hay was later fed to livestock. Erosion of the long bones and other changes similar to those produced by selenium in cereals were noted. The selenium content of the barley was not determined. The other test involved daily drenching of 2 yearling calves with sodium molybdate. One animal weighing 300 lb. received 50 mg. daily until a total of 9·15 g. of molybdenum as sodium molybdate had been given. Another animal of approximately the same weight received 100 mg. doses until 18·3 g. of molybdenum had been administered. The animal receiving the smaller amount tolerated it very satisfactorily. The large quantity caused the other animal to develop pica. It also lost weight and an autopsy is said to have revealed numerous pathological changes not described. While taking into account the small number of experimental animals Beath *et al.* nevertheless considered it of interest that molybdenum in the form of a constituent of barley hay caused much more extensive lesions of the internal organs in an animal than when given as an inorganic drench. It must be pointed out, however, that, although lack of information prevents an exact calculation of the amount of molybdenum consumed in the hay experiment, it would appear to be greater than the amount of inorganic molybdenum consumed in the second experiment. A comparison of the toxicity of inorganic selenium compounds and other inorganic compounds has shown that, in the forms investigated, selenium is more toxic to rats than arsenic, vanadium, tellurium or molybdenum. Further, selenium was the only one of these elements which caused anaemia at the levels investigated (Franke and Moxon, 1937 ; Moxon, 1937). There is also some evidence that the feeding of arsenic in some forms prevents the characteristic symptoms of poisoning in rats caused by feeding seleniferous grain (Moxon, 1938 ; Du Bois, 1940 ; Moxon, 1941).

The experimental production of selenium poisoning in farm animals by means of inorganic selenium has been reported (Miller and Schoening, 1938; Miller and Williams, 1940,[1] 1940 [2]). Miller and Schoening's (1938) experiments showed that symptoms and lesions produced in pigs by the ingestion of sodium selenite were similar to those seen in affected swine in the localities where "alkali disease" prevails and to those produced by feeding toxic grain outside the area as reported by Schoening (1936).

Miller and Williams (1940 [1]) found that, with a single dose of sodium selenite administered orally or as a drench, the minimum lethal doses for horses, cows or calves, and for pigs were 1·5, 4·5 to 5 and 6 to 8 mg. selenium per kg. bodyweight, respectively. There was some evidence that animals in good condition might be relatively more susceptible to the action of selenium than comparatively thin animals. The principal symptoms produced in 2 horses (Miller and Williams, 1940 [2]) by repeated dosing with sodium selenite, emaciation in spite of fairly good appetite, listlessness, looseness of the hair of the mane and tail, softening and scaling of the horny wall of the hoof, were not so severe as those described in cases of chronic poisoning occurring under natural conditions. The lesions observed in the heart, liver, spleen and kidneys at autopsy resembled, to a large extent, those described in field cases. The dose began as 24 p.p.m. of selenium in the feed, was given as a drench after the second month, was doubled at the end of 6 months and quadrupled at the end of 15 months, the animals dying at the end of 17 to 17½ months.

Composition of tissues and fluids.—Rats fed seleniferous grains or inorganic selenium have been shown to store selenium in their tissues. Dudley (1936), in experimental acute selenium poisoning produced in a pig and calf by feeding sodium selenite and in a sheep by giving plants with a high concentration of selenium, found appreciable amounts of selenium in the blood, liver, kidney and spleen. Heart, lungs, brain and muscle contained smaller amounts and no selenium was found in bone and hoof. Similar organs and blood of control animals were free from detectable amounts of selenium. The values found for the sheep fed mixed seleniferous plants containing 1459 p.p.m. of selenium were liver 20, kidney 8, blood 7, bile and spleen 4, lungs 1 p.p.m. of selenium. This animal died in 20 hours.

In experimental chronic selenium poisoning produced in pigs and a horse by feeding sodium selenite, examination of tissues showed similar deposition of selenium except in spleen, blood, bone and hoof. In the chronic cases there was less selenium in the spleen and blood than in acute cases, but there were appreciable amounts in bone and hoof. A horse suffering from "alkali disease" showed 2·5 and 8·0 p.p.m. of selenium respectively, in bone and hoof, and the corresponding values for an affected cow were 1·0 and 5·0 p.p.m. The concentrations of selenium in the bile and urine of both acute and chronic cases indicated that excretion is mainly by the hepatic and renal routes. Selenium has also been found in the excreta of horses in a seleniferous area (Knight and Beath, 1937). Dudley showed that the selenium in the blood of a horse fed sodium selenite (chronic case) was held in the corpuscles and not in the plasma, in the form of a protein-like selenium complex. The presence of selenium in the liver, spleen and kidney of a cow suffering from "frozen feet" in Saskatchewan supported the diagnosis of selenium poisoning based on symptoms and gross pathology (Davidson, 1940). Moxon and Rhian (1943) report average selenium concentrations of 3·05, 2·2, 1·2 and 0·88 p.p.m. fresh weight of hoof, kidney, liver and muscle of 8 steers that had been on an "affected" range for two seasons. Average values for 30 steers that had been on the same range for three seasons were liver 5·7, kidney 4·1 and muscle 3·0 p.p.m. of selenium.

It has been mentioned earlier that sucking young may be affected and the presence of 0·3 to 1·2 p.p.m. of selenium in samples of milk from cows in affected areas has been demonstrated (Byers, 1936; Williams *et al.*, 1940).

Composition of the Soil and Vegetation

Soil.—According to Williams (1938), in most cases the formations from which toxic soils are derived contain less than 10 p.p.m. of selenium. In general, soils in affected areas in U.S.A. and Canada contain between 1 and 6 p.p.m. of selenium in the top 8 inches but much higher values have been found (Byers, 1935, 1937, 1938; Williams *et al.*, 1941). It has been shown, however, that soils of Cretaceous origin in Puerto Rico and the volcanic soils of Hawaii contain considerable amounts of selenium, although they do not give rise to toxic vegetation. Hawaiian samples containing up to 15 p.p.m. of selenium in the surface soil and as much as 10 p.p.m. at a lower level have been reported (Lakin *et al.*, 1938; Byers *et al.*, 1938). The selenium in Hawaiian soils appears to be deposited by rain, the higher the rainfall the higher the selenium content of the soil (Byers *et al.*, 1936, 1938), while in the arid regions of the Great Plains the selenium is present in the parent rock and rain tends to wash it out of the soil (Beath *et al.*, 1934; Franke *et al.*, 1934; Knight and Beath, 1937; Moxon, 1937; Williams *et al.*, 1941; Byers *et al.*, 1935, 1936, 1938).

Vegetation.—The discovery by Robinson (1933) of selenium in toxic grain was followed by investigations into the selenium content of vegetation in affected areas. These investigations showed

that under comparable conditions different plants absorb different amounts of selenium. The principal seleniferous plants were weeds such as *Astragalus bisulcatus, pectinatus* and *racemosus, Stanleya pinnata* and *bipinnata, Aplopappus fremontii* and *Aster parryi*. These plants absorb selenium readily from the soil and more than 4000 p.p.m. D.M. of selenium have been found. On adjacent non-seleniferous soils these plants are wholly absent or very rare and are, therefore, known as " indicator " plants. They are usually avoided by native livestock, but stock brought in from other districts may eat them in times of drought or because of overgrazing. Native grasses, on the other hand, do not readily absorb selenium. In general, they contain less than 4 p.p.m. except under specially favourable circumstances, which will be discussed later, when some of them may contain up to 80 p.p.m. Plants which absorb moderate amounts of selenium include the common cereals and several native plants (Byers *et al.*, 1935, 1936, 1938 ; Moxon *et al.*, 1939 ; Beath *et al.*, 1934,[1, 2] 1935, 1937 ; Knight and Beath, 1937 ; Miller and Byers, 1937 ; Byers and Lakin, 1939 ; Williams *et al.*, 1940, 1941).

Samples of vegetation taken from an area in Wyoming where acute and chronic poisoning were prevalent showed 4 p.p.m. of selenium in mixed grasses and 320 to 4450 p.p.m. in *Astragalus* sp. In a small Wyoming gulch where several thousand sheep died of forage poisoning within a few years, samples of mixed grasses and *Astragalus* contained, respectively, 10 and 4420 p.p.m. of selenium (Byers, 1935).

In Colorado, where " alkali disease " and acute poisoning in cattle occur on an outcrop of Morrison shale, samples of *Astragalus* sp. contained up to 2320 p.p.m. In a 320 acre gulch on upper Niobrara, also in Colorado, where 197 out of 200 sheep driven in died overnight, and where in another year 71 out of 157 sheep died overnight, samples of woody aster, *Astragalus racemosus* and lambsquarters contained respectively 1750, 680 and 890 p.p.m. of selenium. There was scarcely any other kind of vegetation (Byers, 1938). In Saskatchewan in Canada, where the chronic type of poisoning in horses and cattle was observed, a sample of *A. pectinatus* contained 1150 p.p.m. of selenium (Williams *et al.*, 1941).

In Teton county, Montana, where chronic selenium poisoning is believed to be more common than previously recognised, 60 per cent. of the vegetation examined contained over 50 p.p.m. of selenium. In the Irapuato area of Mexico " soliman disease," which is similar to " alkali disease," occurs on vegetation containing 1 to 120 p.p.m. of selenium. It is reported that a drover lost an entire herd of 126 cattle rested for one night in an alfalfa field. A sample of the alfalfa being eaten by a cow suffering from selenium poisoning contained 18 p.p.m. of selenium (Williams *et al.*, 1940).

According to Knight and Beath (1937) and Williams *et al.* (1941) the chronic condition is caused by the daily ingestion of cereals, grasses and other forage plants containing small amounts of selenium. About 4 p.p.m. appears to be the tolerance limit for livestock. The acute poisoning is the result of ingesting large amounts of selenium from the seleniferous weeds already listed. It should be pointed out, however, that poisoning by *Astragalus* plants which contain little or no selenium has been reported, *Astralagus earlei* by Fraps and Carlyle (1936) and *A. pervelutinus wootoni* and *earlei* (Williams *et al.*, 1940), but the symptoms obtained in field and laboratory by feeding these toxic species are not those of chronic selenium poisoning. *A. bisulcatus* also is said to be toxic even when containing no selenium (Beath *et al.*, 1935).

In a study of selenium in Wyoming soils and vegetation, Beath *et al.* (1935) found that most plants on Cretaceous shales absorb molybdenum in varying amounts. Certain plant species, particularly *Oonopsis condensata* and *Xylorhiza parryi* contain appreciable quantities, one sample of *Oonopsis* containing 317 p.p.m. of molybdenum, the roots of the same plant containing 124 p.p.m. The suggestion that any effect of the molybdenum would be obscured by the selenium in this region has neither been supported nor invalidated by the feeding tests reported above.

Several factors appear to affect the selenium content of vegetation. Investigations indicate that selenium may exist in several forms in the soil ; in the form of selenates it is readily available to plants ; in the form of selenites it is relatively unavailable to common crop plants but is available to the seleniferous weeds. These plants are apparently capable of converting the selenium into a form which, when returned to the soil as humus, is available to common crop plants and are therefore known as " converter " plants. Grasses and crops grown in proximity to seleniferous weeds tend to have unusually high selenium contents (Beath *et al.*, 1934, 1935 ; Knight and Beath, 1937 ; Moxon *et al.*, 1939). Olson and Moxon's (1939) experimental work indicates that the availability of selenium to crop plants is dependent on the amount of water-soluble selenium, which in turn depends on or is correlated with the amount of selenium in the organic fraction or humus of the soil. According to Williams *et al.* (1940) the most likely form of insoluble selenium in mature soils is basic ferric selenite and they point out that the Hawaiian soils, high in selenium, which did not produce toxic vegetation were highly ferruginous.

It has been shown that irrigation drainage waters remove soluble selenium from soils that contain it, and the irrigated soils examined, although containing appreciable amounts of total selenium,

produced plants of low selenium content except in Mexico where the water used for irrigation contained selenium. Similarly, the low selenium content of plants on humid soils containing considerable total amounts of selenium is attributed to removal of soluble selenium by percolating water. Seleniferous soils capable of producing toxic vegetation are apparently confined to areas of low rainfall, 20 inches or less per annum (Byers, 1935, 1937, 1938; Williams, 1938; Williams *et al.*, 1940). Yet evidence on the effect of variations in rainfall on the selenium content of vegetation in areas where seleniferous vegetation occurs is conflicting and inconclusive (Byers, 1935; Franke and Painter, 1937).

Accumulations of soluble selenium at various levels in a soil horizon is another possible cause of variation in the selenium content of the plants (Beath *et al.*, 1935; Byers *et al.*, 1935, 1938).

Variations in selenium content with stage of growth of plants have been demonstrated (Byers and Knight, 1935; Beath *et al.*, 1937; Moxon *et al.*, 1939). The stage of maximum selenium content appears to be different for different plants (Beath *et al.*, 1937); the selenium content of sweet clover (*Melilotus officinalis*) increases until the blooming stage and then decreases, and that of western wheatgrass (*A. swithii*) tends to decrease in the later stages of growth (Moxon *et al.*, 1939). The selenium content appears also to increase with increased size of foliage and roots (Beath *et al.*, 1937). Variations have been observed in the selenium content of different parts of the same plant (Beath *et al.*, 1937; Byers *et al.*, 1936, 1938; Lakin *et al.*, 1938; Moxon *et al.*, 1939).

Addition of sulphur appears to inhibit absorption of selenium from artificially selenised soil (Hurd-Karrer, 1934, 1935, 1937; Hurd-Karrer and Kennedy, 1936); but in naturally seleniferous soil, sulphur application does not affect absorption of selenium (Knight and Beath, 1937; Franke and Painter, 1937; Williams *et al.*, 1940) and the water-soluble sulphur content of these soils is of little or no significance in determining the availability of selenium to plants (Olson and Moxon, 1939).

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CHAPTER IX

DISEASES ATTRIBUTED OR ATTRIBUTABLE TO MOLYBDENUM EXCESS

INTRODUCTION AND DISCUSSION

The discovery that large amounts of molybdenum in pasture is harmful to grazing cattle and sheep was made by Ferguson and his co-workers a few years ago. They found that the "teart" pastures of Somerset which caused severe and persistent scouring in cattle and sheep were distinguished from healthy pastures only by their abnormally high content of molybdenum, which ranged from about 20 to 100 p.p.m. D.M. compared with less than 5 p.p.m. in healthy pasture. Further investigation showed that the incidence of the disease was closely related to the amount of water-soluble molybdenum in the pasture. The relationship between a high intake of molybdenum and the incidence of the disease was confirmed by the experimental production of scouring by administration to healthy cattle of sodium molybdate and indirectly by topdressing healthy pastures with sodium molybdate.

Investigations of the soils in affected areas showed that they were derived from the Lower Lias formation and that the uptake of molybdenum, and hence the teartness of the pastures, was greatest on molybdenum rich soils which were alkaline and calcareous. Application of sulphate of ammonia to the soil reduced the teartness of the herbage.

The mode of action of molybdenum is not known, but the clinical picture of molybdenum poisoning and the effectiveness of copper sulphate as an antidote suggest some interference with copper metabolism (see p. 51).

It has been suggested that in some parts of America a high content of molybdenum in certain seleniferous weeds complicates the picture of alkali disease, but the evidence in support of this view is not conclusive (see p. 77). Molybdenum excess has also been suggested as the cause of the bovine haematuria enzootic in some parts of Australia. In these areas samples of capeweed and meadow hay contained up to 8 p.p.m. D.M. of molybdenum compared with less than 1 p.p.m. in control areas, and animals depastured in the affected areas showed abnormally high levels of molybdenum in liver, faeces and urine. Applications of gypsum to the soil reduced the uptake of molybdenum by the pasture, and it is of interest to note that bovine haematuria in British Columbia is said to be controlled by topdressing with gypsum.

Mitchell and his co-workers have recently made the interesting observation that in certain areas in Easter Ross, Scotland, topdressing pasture with 2 lb. cobalt chloride per acre to remedy a deficiency of cobalt increased the molybdenum content from 6 to 9 p.p.m., which they considered approaching teartness levels.

The scouring disease of Somerset has been described and discussed in the section dealing with copper deficiency. The following is a brief account of the investigations into the etiology of bovine haematuria.

ENZOOTIC HAEMATURIA OF CATTLE

The occurrence of bovine haematuria, or "redwater," of a non-infectious type has been investigated in Australia, British Columbia, New Zealand and Italy. Hill *et al.* (1933), in British Columbia, and Gottardi (1935), in Italy, observed that the condition occurred most frequently on acid soils and infertile soils. In a more recent study in British Columbia, Bankier (1943) found no relation between the occurrence of the disease and soil type, and preliminary analyses showed no significant difference in manganese, sodium, potassium, calcium, magnesium, aluminium, iron, cobalt or selenium content of "redwater" and non-"redwater" soils.

The only significant discovery so far has been that of the Australian workers (C.S.I.R. Ann Rep., 1940) who found that the pasture in affected areas consistently contained higher than normal amounts of molybdenum although much less than the "teart" pastures of Somerset.

They found that application of gypsum to the soil of the affected areas resulted in a marked decrease in the uptake of molybdenum by the pasture, and it is of interest to observe that in British Columbia topdressing the land with gypsum appears to have some value in controlling the disease (Bankier, 1943). The Australian workers found that cows depastured in the affected areas showed an abnormally high storage of molybdenum in the liver while faeces and urine also contained considerable amounts. Attempts to produce haematuria experimentally by feeding molybdenum in amounts equivalent to that provided by the Australian pasture where haematuria is enzootic have been begun. Some analytical data on the molybdenum content of these Australian pastures given in a private communication (Bull, 1943) are shown in the table below. As far as possible samples consisted of capeweed, which was considered a good index plant.

Haematuria Areas		Control Areas	
	Mo p.p.m. D.M.		Mo p.p.m. D.M.
Mature plant (no manure) . .	8·0		
Immature plant (superphosphate) .	1·33	Capeweed	0·46
Immature plant (gypsum) . .	0·44	,,	0·37
Meadow hay (no manure) . .	3·2	—	—
Meadow hay (gypsum) . . .	1·0	—	—
Capeweed	5·4	—	—
,, ...	4·76	—	—

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CHAPTER X

MANGANESE EXCESS

"FORAGE ANAEMIA" ASSOCIATED WITH INFECTIOUS ANAEMIA

Discussion

The possibility that horses may suffer from an excess of manganese in pasture and hay has only recently been suggested. The discovery arose from investigations of infectious anaemia in horses, which is an important economic problem on natural forest pastures of Scandinavia and Finland. Svanberg found that pasture and hay from affected areas were high in manganese but there was no clear correlation between the manganese content and the incidence of infectious anaemia. Affected

horses showed oedema and emaciation in addition to signs of anaemia and fever, but sometimes horses which were not anaemic and had no fever showed oedema, emaciation and ataxia. This associated condition, which they called " forage anaemia " to distinguish it from the infectious disease, they attributed to deficiency of vitamin B_1 since it was found that its occurrence could be prevented by administration of yeast, the symptoms could be reproduced artificially on a diet deficient in the B vitamins, and limited analytical data indicated a deficiency of vitamin B_1 in hay from " affected " areas. A connecting link between these two conditions was established by the discovery that horses on the vitamin B deficient diet showed an increased susceptibility to infection with the anaemia virus, which was reduced by administration of yeast. Carlström and Hjärre (1938) point out that vitamin B_1 disappears more rapidly from " affected " Norrland hay than from normal hay, and suggest that the vitamin is destroyed by oxidation in the presence of large amounts of manganese. They consider it possible, however, that an absolute deficiency of vitamin B_1 in hay may predispose towards infectious anaemia in the absence of excess manganese.

A direct effect of a high intake of manganese on the vitamin B_1 metabolism of the animal has not been suggested by these workers, but it would appear from the rat experiments of Sandberg and his co-workers that such a possibility should not be excluded. Perla and Sandberg (1939) showed that administration of manganese had a neutralising effect on toxic doses of vitamin B_1 and that administration of vitamin B_1 minimised the effect of a toxic dose of manganese. Further, Sandberg *et al.* (1939) showed that administration of manganese to rats on a vitamin B_1 deficient diet reduced the time required for vitamin B_1 depletion and resulted in an abnormal retention of manganese. The retention of manganese was reduced when large doses of vitamin B_1 were given. The production of some of the symptoms of " forage anaemia " in 1 of 2 horses fed normal fodder with the addition of twice the amount of manganese found in " affected " hay, the lack of symptoms in horses fed manganese in an amount equivalent to that found in " affected " hay and the fact that these horses were not as susceptible to infection with the anaemia virus as animals fed " affected " hay may possibly be accounted for on the basis that manganese in fodder containing an adequate supply of vitamin B_1 is not toxic at the same level as in hay which is deficient in vitamin B_1. Further investigations are required, however, to establish the relationship between manganese excess and vitamin B_1 deficiency in horses.

There appear to be several factors which contribute to the high manganese content of these natural forest pastures. Uncultivated soils usually contain more manganese than cultivated soils, the acidity of the soil makes the manganese more available to the plants and the high manganese content of certain forest trees may also be concerned. Liming reduces the assimilation of manganese by the plant, but very heavy liming would probably be required to prevent anaemia in horses on these forest pastures.

Occurrence and Description of the Disease

Infectious anaemia has been reported from almost every country in the world since it was first described as an independent disease by Lignée in 1843 (Hutyra and Marek, 1926). It was early recognised that the disease was caused by an ultrafiltrable virus, but it also appeared from the localised nature of its occurrence and the difficulties involved in infecting animals from healthy areas with doses of infected serum that the incidence of the disease depends to some extent on the quality of the fodder. This aspect of the disease has recently been investigated in Scandinavia and Finland, and comes within the scope of this review.

Infectious anaemia is an extensive and important economic problem in horses grazing or fed on hay from the natural forest pastures of Norway, N. Sweden and N. and E. Finland. It was first described in Sweden by Ekvall (1895). Horses show signs of anaemia, pallor of the mucous membranes, decreased blood volume and low red cell counts. The pulse rate is high and there is fever, continuously or intermittently. Oedema and emaciation occur in spite of good appetite and the disease usually ends fatally, acute cases dying within a few days of the onset of the disease and chronic cases after periodical recovery and relapse. *Post mortem* examination of the liver shows fatty infiltration, deposits of fat and haemosiderin in the Kupffer cells and reticulo-endothelial proliferation.

In areas where infectious anaemia is prevalent horses may show oedema, ataxia, emaciation and fatty infiltration and haemosiderosis of the liver without necessarily suffering from infectious anaemia. This condition, which has been called " forage anaemia," although anaemia is not necessarily present, is distinguished from infectious anaemia by lack of fever and the absence of endothelial proliferation (Carlström and Hjärre, 1938). A similar condition not necessarily associated with infectious anaemia has been observed in the infectious anaemia districts of N. and E. Finland (Hällfors, 1933).

Experimental Studies

Feeding experiments.—Since pasture and hay in the affected areas is frequently very high in manganese an attempt was made (Carlström and Hjärre, 1938) to produce the condition in 4 horses fed normal fodder with manganese added daily for 2 to 4 months in amounts equivalent to that of "affected" pasture or, in the case of 2 of the horses, twice that amount after 2 months. Of the 2 horses receiving the double dose one showed emaciation, slight oedema of the abdomen, staring coat and ataxia by the fourth month, symptoms characteristic of the naturally occurring "forage anaemia." *Post mortem* examination showed only slight cirrhosis of the liver in the affected horse, although metabolism experiments and analyses of organs showed that these horses retained more manganese than horses receiving hay rich in manganese. The other horses were not affected. Manganese did not therefore appear to be the direct cause of the disease. Controlled infection experiments were made with filtered serum of infected horses. Administration of manganese had a slight protective effect or none, but horses fed on "affected" hay had a reduced resistance. The possibility of vitamin deficiency was next explored, and this field gave more promising results. Horses fed on a vitamin B deficient diet of polished rice, straw, sodium phosphate, chalk and cod liver oil showed emaciation, oedema, ataxia, fatty infiltration of liver cells, and deposits of fat and iron in the Kupffer cells, like horses grazing "affected" pasture. It was found that the administration of 50 to 100 g. yeast daily to horses fed the vitamin B deficient diet and to horses fed "affected" hay prevented the appearance of such symptoms. The resistance to the virus infection of horses fed the vitamin B deficient diet was low and addition of yeast protected also against infection. Daily administration of 1 g. ascorbic acid had no protective effect. It is concluded that horses in the anaemia districts suffer from a deficiency of vitamin B in the fodder, "forage anaemia," which lowers their resistance to the action of the anaemia virus.

Composition of the Soil and Pasture

The soil in affected areas is usually acid with a high sulphur and low calcium content. The soil is also high in manganese. The calcium and manganese contents of "affected" and unaffected soil given by Svanberg (1938) are shown in Table I. Svanberg suggests that the ratio of calcium to manganese in the soil may be important.

TABLE I

The Calcium and Manganese Content of Healthy and "Affected" Soils

	No. of Samples	CaO per cent. D.M.	MnO per cent. D.M.	$\frac{MnO}{CaO}$
Anaemia free district	25	0·70	0·067 (0·022–0·17)	0·10
"Anaemia district," but with no actual cases	7	1·38	0·33 (0·059–1·2)	0·24
"Anaemia district," with cases of anaemia .	24	0·40	0·20 (0·019–1·8)	0·50

Pasture.—A large number of determinations of the manganese, iron, copper, calcium, phosphorus, potassium, sodium and nitrogen contents of pasture and hay from areas in Norway, Sweden and Finland where the disease is endemic and from unaffected areas have been reported (Svanberg, 1934, 1938; Svanberg *et al.*, 1935). Low values for calcium, phosphorus, sodium and chlorine were found, but there was no correlation between the occurrence of anaemia and deficiency of any one of these elements in the forage. The mean values for copper oxide were 15 and 19 p.p.m. (air dry basis) in anaemia and anaemia-free areas, with minimum values of 4 and 6 p.p.m. of copper oxide respectively. Svanberg (1935) suggested that further attention should be given to this in the anaemia areas despite the absence of obvious correlation with the disease. The most outstanding feature of the pasture and hay was the high manganese content. There was no clear correlation between the manganese content of the vegetation and the incidence of anaemia (Table II), and Svanberg (1938) suggested that the high manganese content might be an indicator of conditions which produced some other abnormality in the vegetation.

Carlström and Hjärre (1938) investigated the possibility of deficiency of vitamin B_1 and showed that "affected" Norrland hay contained from 0 to 300 I.U. vitamin B_1 per kg. compared with 500 to 800 I.U. for good timothy hay and 700 to 1000 I.U. for good clover hay. They found that vitamin B_1

TABLE II

MANGANESE CONTENT OF HEALTHY AND "AFFECTED" VEGETATION

	No. of Samples	Average MnO per cent. Air Dry Basis	Percentage Distribution of Samples *				
			1	2	3	4	5
Hay from ploughed land							
Anaemia-free district	75	0·0057	89	11	0	0	0
"Anaemia district" with no actual cases	21	0·0166	38	57	5	0	0
"Anaemia district" with cases	35	0·0164	40	54	3	0	0
Uncultivated vegetation and hay							
Anaemia-free district	57	0·0344	4	68	24	4	0
"Anaemia district" with no actual cases	24	0·0443	4	54	25	17	0
"Anaemia district" with cases	346	0·0642	1	35	38	26	1

* 1 = 0·001 — 0·0099 % MnO
2 = 0·01 — 0·039 „
3 = 0·04 — 0·079 „
4 = 0·08 — 0·199 „
5 = 0·2 —

disappeared much more rapidly from Norrland hay than from normal hay and suggested that this was probably due to oxidation in the presence of large amounts of manganese. They also suggested that an absolute deficiency of vitamin B_1 in hay may predispose towards infectious anaemia in the absence of excess of manganese.

The factors affecting the manganese content of soil and vegetation have recently been reviewed by the Imperial Bureau of Soil Science (1940) and the results briefly summarised are as follows:

Cultivated soils usually contain less manganese than uncultivated soils.

The solubility of manganese in the soil depends largely on the soil reaction and the oxidation-reduction equilibrium.

Manganese is one of the most easily exchangeable bases, particularly under acid conditions.

Liming reduces the availability or solubility of manganese presumably as a result of oxidation.

The manganese of different plant species grown on the same soil varies considerably.

Certain forest trees, particularly firs, birches and elms contain rather large amounts of manganese.

Liming, which reduces the amount of soluble manganese in the soil, sometimes causes "grey speck" disease in oats which has been attributed to deficiency of manganese.

An interesting observation is that the use of copper in the control of "reclamation disease" frequently causes "grey speck" to appear in oats.

Svanberg's observations in Sweden confirm that pasture on ploughed land has a lower manganese content than that of pasture on uncultivated land of the same *p*H, and that the manganese content of uncultivated pasture is correlated with the *p*H of the soil. He observes that although liming reduces the manganese assimilation by the plant, very heavy liming would be required to prevent anaemia in horses in Northern Sweden.

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CHAPTER XI

FLUOROSIS

Fluorine poisoning occurs in domestic animals in areas such as Morocco and Tunis, where there are large natural deposits of phosphate ; it has also been reported to occur after local volcanic eruptions as in Iceland and in the neighbourhood of chemical industries which utilise materials containing fluorine.

It is not proposed to discuss the subject here in detail because the indications are that the fluorine content of the pasture is not important in the etiology of the disease. Velu (1933) reported that fluorine loses its toxicity when " fixed " by vegetation, e.g., lucerne. Gaud *et al.* (1934) analysed the leaves, stem and roots of fig trees and the grain and straw of wheat and barley, and found that in fluorisis or " darmous " areas of Morocco the fluorine content of these plants was about 10 times that of non-fluorosis areas. Feeding experiments with guineapigs indicated that the fluorine supplied by a ration of barley and carrots was a more important cause of fluorosis than the drinking water, but subsequently it was shown that $\frac{3}{4}$ of the fluorine supplied by the food was present in the dust contaminating it. Pierce (1939) quotes experiments which showed that simultaneous ingestion of green grass reduced the toxicity of fluorine for pigs and rats.

Hart *et al.* (1934) have shown that application of phosphatic fertilisers containing fluorine in plot experiments over a period of 16 to 36 years had no consistent influence on the fluorine content of crops, and there were indications that a considerable portion of the applied fluorine may be carried away from the soil in the drainage water.

Reid (1936) found that in general the fluorine content of foods grown near a fluorite mining area was not influenced by the fluorine content of the soil. The work of Machle *et al.* (1939) indicates that there may be no direct correlation between food fluorine and water fluorine, and that the former is relatively unimportant as a cause of human mottled enamel. Green (1941), discussing an outbreak of industrial fluorosis which occurred among animals in Britain, states that although pasture grass and hay were definitely incriminated, the high values observed for fluorine in the vegetation arose by surface contamination and not by absorption through the roots from local soil naturally high in fluorine.

For further information and an excellent bibliography on the wider aspects of this subject, the reader is referred to the recent review by Pierce (1939).

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ARSENIC POISONING

The occurrence of an unusual ailment in livestock resulting in the death of cattle, especially dairy cows, on drained swamp land at Reporoa in New Zealand, was found to be associated with the high arsenic content of the soil and drinking water. With the exception of one sample, however, the pasture did not show an abnormal content of arsenic, and the disease was attributed to ingestion of soil and water rich in arsenic.

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CHAPTER XII

NITRATE EXCESS

"OAT HAY POISONING"

Discussion

Serious outbreaks of poisoning in ruminants, particularly in cattle, have been reported, following ingestion of oat hay or straw containing between 3 and 7 per cent. of potassium nitrate. The disease has been shown to be caused by reduction of the nitrate to nitrite in the rumen, and possibly to some extent before ingestion, with resulting methaemoglobin formation. Although the oat plant seems to be especially liable to develop high concentrations of nitrate, other plants and weeds are sometimes found to contain toxic amounts. Sjollema (1931) has shown in his investigation of "grass tetany" that some Dutch pastures contain as much as 2 per cent. potassium nitrate on a dry matter basis, although there was no evidence of methaemoglobinaemia.

Although not a "mineralosis," oat hay poisoning probably merits brief description here because the nitrate is apparently present almost entirely as potassium nitrate and the conditions governing the potassium nitrate content of plants are not yet fully understood.

Occurrence and Description of the Disease

Outbreaks of "oat-hay poisoning" have been reported, mostly in cattle, in Wyoming, Colorado, Kansas and South Dakota in the U.S.A. and in Saskatchewan in Canada, following ingestion of oat hay or oat straw, in most cases damp or mouldy hay.

Affected animals show acceleration of the pulse, quickened respiration, trembling, weakness, and staggering gait. They cease to eat and soon collapse and die without convulsions. Cyanosis of the tongue and sclera is observed and the blood is of a reddish-brown colour due to the presence of methaemoglobin. Cattle appear to be more susceptible than sheep or horses. (Newson *et al.*, 1937; Thorp, 1938).

Experimental Studies

Feeding experiments.—The condition has been reproduced experimentally in cattle, sheep and horses by feeding potassium nitrate or an extract of toxic hay containing the potassium nitrate. An extract of the hay after removal of the potassium nitrate was not toxic (Bradley *et al.*, 1939, 1940 [1, 2]; Davidson *et al.*, 1941). Methylene blue acts as an antidote and prevents the occurrence of the disease (Bradley *et al.*, 1940 [1]). Bradley *et al.* (1940 [2]) tentatively set 1·5 per cent. potassium nitrate or 0·2 per cent. nitrogen as nitrate as the lower toxic limit.

Composition of the Hay

Samples of affected oat hay showed a high content of potassium nitrate ranging from 3·2 to 7·2 per cent. (Bradley *et al.*, 1939). Doughty and Warder (1942) analysed 65 samples of oat straw collected from a wide range of territory in Saskatchewan and Alberta and found that 32 samples contained 0·2 per cent. or more nitrogen as nitrate. The values varied from 0·003 to 0·556 with a mean of 0·165 per cent. nitrogen as nitrate. According to Davidson *et al.* (1941) the distribution of potassium nitrate in a sample of toxic oat hay was: leaves 4·2, stalks 2·5 and heads 0·7 per cent. Other plants and weeds appear capable of producing the disease but oat hay is the principal offender. The "affected" hay did not contain appreciable amounts of nitrite and since it is nitrite, not nitrate, which causes methaemoglobinaemia, it was assumed that the nitrate was reduced to nitrite in the gastro-intestinal tract of the animal. This view was supported by the demonstration of nitrite in the bile and urine of affected animals (Bradley *et al.*, 1939) and by the experiments of Seekles and Sjollema (1932), who introduced potassium nitrate into the rumen of cattle and got symptoms of nitrite poisoning and evidence that 10 per cent. of the nitrate was changed to nitrite in the rumen. According to Davidson *et al.* (1941) the nitrate content of extracts of oat straw decreases very rapidly over a few days, while the nitrite content increases. Olson and Moxon (1942) also found in preliminary experiments with oat hay and redroot (*Amaranthus retroflexus*), and in more detailed work with redroot, that reduction of nitrate to nitrite takes place *in vitro* under varied conditions of

temperature when the material is wet. They isolated from the redroot an organism, *Brucellus subtilis*, capable of reducing nitrate to nitrite, and suggested that this is also the reducing agent in the rumen. The work of Rimington and Quin (1933), who showed that poisoning in sheep in South Africa after ingestion of species of *Tribulus* was due to the high nitrate content of the plants, is of interest in this connection because their results indicated that, under certain field conditions, the nitrate in the plant was reduced to nitrite.

Davidson *et al.* (1941) report that the potassium nitrate content of many plants is high during the prebloom stage, but rapidly diminishes at or after blooming. Analyses of samples of oat straw indicated that drought favoured a low nitrate content, but Doughty and Warder (1942) found in plot experiments with oat straw that drought during the heading and ripening period increased the nitrate content. These workers also found that crops grown on summer-fallow land contained more nitrate than crops grown on stubble land, and applications of potassium nitrate to the soil increased the nitrate content of oat straw grown on it.

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CHAPTER XIII

GENERAL DISCUSSION

Mineral deficiencies

In a recent review of the significance of trace elements in nutrition Underwood (1940) has pointed out that there are now no less than 13 mineral elements known to be essential for animal life. These are calcium, phosphorus, magnesium, sodium, potassium, chlorine, sulphur, iron, copper, manganese, zinc, iodine and cobalt. The present study has been confined to those minerals of which naturally occurring deficiency in grazing animals is known or suspected.

The bridging of the gap between suspicion and established fact is difficult. There is no known simple rule by which to decide when a state of deficiency exists. The level of minerals in the blood is not always a reliable guide. Low levels of inorganic phosphorus in the blood are apparently characteristic of dietary phosphorus deficiency in ruminants, but low levels of serum calcium in grazing animals appear, in most cases, to be the result of endocrine disturbances rather than dietary calcium deficiency. The same may be true of serum magnesium. Nor are the levels of cobalt and copper in blood reliable indices of the supply. The blood appears to draw upon reserves in the liver and other organs which are themselves a better index of deficiency of these elements.

Pica or depraved appetite is usually an indication of deficiency. When this takes the form of osteophagia, or a craving for bones, phosphorus deficiency is indicated, but in other picas the real deficiency may not be apparent from analyses of the materials eaten. For instance, animals may appear to seek earths or salts high in sodium or iron or consisting mainly of silicates while their real objective may be some trace element present.

Feeding tests with purified salts of single minerals are more reliable in the diagnosis of deficiencies, but are not infallible. For example, in grass tetany the feeding of magnesium may compensate for a temporary inability of the animal to mobilise magnesium from the skeleton and need not necessarily indicate an absolute deficiency of magnesium. The experimental production of deficiency disease by means of diets adequate in all respects other than the specific mineral under investigation

is useful as a method of defining the symptoms of a particular deficiency, but such experiments are not often made with ruminants.

The combined evidence of feeding experiments and measurement of reserves show that deficiencies of phosphorus, cobalt, copper and iodine occur among grazing animals. In areas where signs of deficiency diseases are observed the pasture would be expected to have an abnormally low content of the mineral involved. Where this is found to be so, the case for mineral deficiency is proved, but even if the pasture appears adequately supplied, it is not disproved. The possibility that some factor affects the availability to the animal of the mineral in question remains to be considered.

There is ample evidence that aphosphorosis in grazing cattle is due to deficiency of phosphorus in the pasture, but the question of deficiency of minor elements is complicated by several factors :

1. Difficulties of analysis.
2. Seasonal variations in the composition of pasture.
3. Contamination of samples with soil and the problem of how much of the minerals ingested in the soil is utilised by the animal.
4. The use of supplementary licks which may contain traces of the element in question. For instance, in certain parts of Sweden the use of phosphatic supplements which contain copper may obscure a correlation between the copper content of the pasture and the incidence of copper deficiency in livestock.

In general, however, it may be said of those areas where pasture analyses have been made that cobalt deficiency in livestock is due to deficiency of cobalt in the pasture. In certain areas this is also true of copper deficiency, but in other areas the problem appears to be one of availability of the pasture copper. The position with regard to iodine is not clear, but it seems likely that factors other than the iodine content of the pasture contribute to the cause of goitre in grazing animals. Analyses of pasture for sodium and chlorine, taken in conjunction with the evidence of experimental sodium and chlorine deficiency, indicate that deficiency of sodium and chlorine with respect to the requirements of growth and health may occur in dry or semi-arid countries, but, in general, pastures supply the needs of all livestock, except high milking cows, for sodium, and all the needs of livestock for chlorine.

There does not appear to be any evidence of deficiency of magnesium in pasture, but the issue is confused by the fact that most published analyses do not take into account that manganese, if present in sufficient quantities, may give erroneously high values for magnesium. There is no evidence that livestock suffer from naturally occurring deficiencies of calcium, iron, manganese, potassium or zinc in pasture, although a conditioned deficiency of calcium due to lack of vitamin D may occur. Sulphur deficiency has not been discussed since the question is one of deficiency of protein, and especially of essential amino-acids containing sulphur.

Although nickel is not included in Underwood's list of essential elements, there is some evidence (Filmer and Underwood, 1936, 1937 ; Dixon, 1936, 1937) that nickel exerts a supplementary beneficial effect when fed along with suboptimal amounts of cobalt to animals suffering from deficiency of cobalt.

Mineral excesses

Livestock disease may occur from ingestion of pasture or hay containing abnormally large quantities of some minerals. Sometimes there are indications that excess of one element interferes with the absorption or utilisation of another so that in effect a deficiency state results. This may be the case in " swayback " of lambs in Derbyshire, where the high calcium, iron, lead or zinc content of the pasture may interfere with the absorption of copper to produce a copper deficiency state where there is no abnormality in the copper content of the pasture. It has also been suggested that anomalies in the copper deficient areas of Australia may be explained on the basis of variations in availability of pasture copper. Another possible example is the " scouring disease " on the " teart " pastures of Somerset which in some ways resembles copper deficiency and is associated with excessive intake of molybdenum from pasture well supplied with copper. There are also indications that excess of manganese in hay or pasture may exert a toxic effect on horses by oxidising the vitamin B_1 in the hay or pasture, thus producing avitaminosis B_1.

An excessive intake of potassium may interfere with calcium, phosphorus and nitrogen absorption, but the theory that excess of potassium over sodium in the pasture is the cause of lecksucht in cattle does not appear to have any sure foundation.

An important toxic element in vegetation is selenium. In this case, however, there has been no suggestion of any indirect effect involving the absorption of other elements. " Oat hay poisoning " appears to be due to the presence in hay or straw of excessive amounts of potassium nitrate, which is converted to nitrite in the rumen, and possibly to some extent also before ingestion.

Fluorine and arsenic poisoning have been discussed very briefly, but soil and water rather than pasture appear to be the main sources of these toxicants.

Svanberg (1941), in a review of trace elements and pasture production in Sweden, states that very high magnesium levels, which occasionally occur in Swedish hay grown on fertile clay and fen soils, are associated with disturbance of water metabolism and greatly reduced milk yield in cattle.

In conclusion, it should be emphasised that despite the extensive literature which has accumulated on mineral deficiency diseases in livestock, there are still many gaps in our knowledge. The more important of these are as follows.

Cobalt feeding is beneficial in nakuruitis but the cobalt reserves of the animals have not yet been determined, nor are there figures for the cobalt content of Nakuru pastures. In the pining areas of Britain, where cobalt feeding has proved beneficial, there is no information about the cobalt reserves of the animals, and with the exception of the Dartmoor and Ross-shire areas the cobalt content of the pastures has not been determined.

Coast disease in Australia is attributed to a dual deficiency of cobalt and copper, since both of these elements must be fed to control the disease, but the cobalt reserves of the animals have not been determined, and for information on the cobalt content of the pastures there is only the value for one Tasmanian sample given in a private communication. Values for the copper content of " coasty " pastures do not seem to have been published, although Dumaresq appears to have found " coasty " pastures on King Island, Tasmania, where falling disease occurs, low in copper, and fertilising with copper and cobalt has been found to control the disease in Tasmania.

A similar position obtains in the salt sick areas of Florida where cobalt and copper feeding is curative. Workers have failed to demonstrate the presence of cobalt spectrographically in the predominant grass in healthy or " affected " pasture, and although the organs and tissues of calves from affected and healthy dams have been analysed for trace elements, including cobalt and copper (without any deficiency being demonstrated) the cobalt and copper reserves of the sick animals have not been determined.

Investigators of European licking diseases who have correlated the disease with the low copper content of the pasture and who have obtained beneficial results with copper feeding, have not confirmed their findings by analysing the animal organs for copper.

It would be of interest also to know if the copper reserves of animals which respond to copper feeding on the " teart " pastures of Somerset are low despite the adequate copper content of these molybdenum rich pastures.

It appears to be fairly well established that goitre results from iodine deficiency, but the exceptions in South Africa and Sweden have still to be explained. Although goitre does not seem to occur on pastures well supplied with iodine, a given level of iodine in pasture or hay may be goitre-producing in one area and healthy in another. Further investigations are needed to establish the factor or factors responsible for such anomalies. In the meantime the iodine content of the thyroid is the most reliable index of the need for iodine administration to livestock.

The licking diseases of Germany and parts of Sweden still remain shrouded in mystery which systematic feeding trials with minerals known to be essential might dispel.

" Oat hay poisoning " has been established as due to nitrite poisoning from hay containing excessive amounts of potassium nitrate, but investigations are still needed to explain the conditions which predispose to abnormal concentrations of potassium nitrate in hay.

The extensive occurrence of infectious anaemia in horses makes it desirable that the Swedish findings be tested in other infected areas. In Europe this may not be possible until after the war, but in view of the great increase in the incidence of the disease after the last war the possibilities of vitamin B_1 therapy and prophylaxis should be kept in view.

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APPENDIX

SUMMARY TABLE OF INVESTIGATIONS

Disease	Species Affected	Country	Principal Areas Investigated	Blood and Tissues	Response to Feeding Tests	Pasture Characteristics	Effect of Specific Fertilisers on Incidence of Disease	Experimental Production of Disease	Conclusions
Bush-sickness	Sheep and Cattle	New Zealand	Volcanic soils of N. Island central plateau. Granite soils of S. Island	Low Co reserve in organs which normally store Co	Co feeding preventive and curative	Low Co content related to incidence of disease: < 0·07 p.p.m. D.M. of Co unhealthy for sheep; < 0·04 for both sheep and cattle	Co fertilising effectively controls the disease	—*	Uncomplicated Co deficiency state due to lack of Co in the pasture
Morton Mains Disease	Sheep, especially lambs	New Zealand	Southland	Ditto	Ditto	Pasture upon which lambs developed the disease, 0·04 to 0·07 p.p.m. D.M. of Co	Ditto	—	Ditto
Enzootic Marasmus	Sheep and Cattle	Australia	W. and S. Australia	Ditto	Ditto	Low Co content in affected areas, 0·02 to 0·06, mean 0·04 p.p.m. D.M.	—	—	Ditto
Nakuruitis	Cattle	Africa	Nakuru, Kenya	—	Co feeding beneficial	—	—	—	Probably Co deficiency
Pining or Pine	Sheep	Britain	Ross-shire Scottish Borders Wales, 1 centre Dartmoor	— — Ditto —	Controlled by Co feeding Ditto Ditto Co + Mn + Zn beneficial	Pasture, 0·08 p.p.m. D.M. of Co. Co content 0·05 to 0·20 p.p.m. D.M. No correlation with disease but seasonal variations may obscure this — Affected areas, 0·20 p.p.m. of Co. Recovery areas, 0·45 p.p.m.	Ditto — — —	— — — —	Probably Co Deficiency
Grand Traverse Disease	Cattle	U.S.A.	Michigan	—	Co feeding curative	"Affected" hay, 0·03 to 0·06 p.p.m. of Co. Healthy hay, 0·12 p.p.m. of Co	—	—	Co deficiency
Coast Disease	Sheep, Cattle, Goats	Australia	Coastal areas of S. and W. Australia and Tasmania	Low Cu reserve in organs which normally store Cu	Co + Cu feeding controls the disease. Neither alone is effective	Sample of affected pasture (Tasmania), 0·10 p.p.m. D.M. of Co (sample slightly contaminated)	Topdressing with Cu + Co effective in controlling the disease in Tasmania	—	Probably dual deficiency of Cu + Co
Salt sick	Cattle, Sheep, Goats	U.S.A.	Florida, sandy soils	—	In some areas Fe and Cu is effective in controlling the disease; in others Co is also required. The Fe supplement probably contained traces of Co in some areas	Failure to demonstrate presence of Co spectrographically in either sick or healthy wire grass suggests method inadequate. No significant difference in Cu content. Range 4·7 to 10 p.p.m. D.M.	—	Feeding experiments with calves indicate that "salt sick" hay may produce Co deficiency uncomplicated by Cu deficiency	Probably Co deficiency usually associated with Cu deficiency. Deficiencies not demonstrated in pasture
Enzootic Ataxia associated with Anaemia . . . . and Stringiness of wool	Lambs Gestating Ewes Adult Sheep	Australia	Coastal and semi-coastal areas of W. Aust., also on calcareous soils of S. Australia	Low Cu reserve in organs which normally store Cu. Low Cu content of ewes' milk	Feeding Cu to gestating ewes prevents anaemia and stringy wool and occurrence of ataxia in the progeny	Values of < 3 p.p.m. D.M. of Cu indicate unsound pasture. Between 3 and 5 are marginal and > 5 are sound. Some anomalous results	Topdressing with Cu effectively controls ataxia	—	Uncomplicated Cu deficiency state in ewes and lambs due to lack of Cu in the pasture eaten by the ewe
Swayback	Lambs	Britain	Derbyshire	Low Cu reserves of ataxic and normal lambs in affected areas	Feeding Cu to gestating ewes or to lambs controls swayback in lambs	Pastures well supplied with Cu, 12 to 27 p.p.m. D.M. High Pb, Ca, Fe and Zn	—	—	Cu deficiency state. Possibly the availability of the pasture Cu is affected
Sudden Death or Falling Disease	Cattle	Australia	South and west coastal areas	Low Cu reserve in liver	Controlled by Cu feeding	Cu usually < 2 p.p.m. D.M.	—	—	Cu deficiency state associated with lack of Cu in pasture
Scouring Disease	Cattle, Goats	Holland	Sandy soil and reclaimed "polders"	Low Cu reserve in organs. Low blood Cu and low Cu content of hair	Cu feeding gave very good results	Cu content of pasture mostly > 5 p.p.m. D.M.; hay 3·6 to 4·65 p.p.m. and low Mn content. Mo not abnormal. No other obvious abnormality	—	—	Probably Cu deficiency state but no absolute deficiency of Cu demonstrated in the pasture
Scouring Disease ("Teart" pastures)	Cattle, Sheep	Britain	Somerset	—	Cu feeding preventive and curative	Pasture well supplied with Cu, 11 to 18 p.p.m. D.M. High content of Mo, 20 to 100 p.p.m. D.M., related to incidence of disease	Manuring healthy herbage with Mo causes scouring in grazing cattle and increase in Mo content of the herbage	The disease has been produced by feeding inorganic salts of Mo to cattle	Direct cause is the abnormal Mo content of the pasture. This may indirectly interfere with Cu metabolism; hence effect of Cu feeding
Licking Diseases †	Cattle, Sheep, Goats	Holland Sweden	Sandy and moor soils especially reclaimed soil Gisselås	—	Ditto Controlled by feeding molasses containing 14 to 16 p.p.m. of Cu	Cu in hay usually between 2 and 3 p.p.m. D.M. Cu in hay 0·5 to 5·2 p.p.m. Average 2 p.p.m.	— —	— —	Cu deficiency due to lack of Cu in pasture Probably ditto
Grass Tetany	Cattle, principally lactating cows	Holland		Low blood Mg; often low serum Ca	Cured by injection of Ca + Mg	Protein and KNO_3 high. K : Na wide, sometimes > 50 : 1. Ca usually rather low. Mg apparently normal	—	Not produced on diets high in protein or in KNO_3	No conclusion regarding etiology of grass tetany can be drawn from the available data
Ditto	Ditto	New Zealand		Mg content of milk, bones and organs of affected cows within normal limits. Blood low in Mg, often high in inorganic P	Ca + Mg therapy gave no useful results but feeding of silage with added dolomite is beneficial	Protein high, P high, Ca low, K : Na ratio normal. Mg considered slightly subnormal	—	—	
Ditto	Ditto	Britain	Lincolnshire	[redacted] blood often low serum Ca, high Mn content of blood	—	High Mn content. Mg apparently normal	[redacted]	Feeding Mn in the amounts which occur in these pastures depressed serum Mg but did not produce tetany	
Ditto	Ditto	Ireland		Low blood Mg, often low serum Ca	—	High Mn content. Mg apparently normal	—	—	
Goitre	Cattle, Sheep Goats, Horses, Pigs	World-wide		Low I content of thyroids	Controlled by feeding I except in S. Africa and Sweden	Limited data indicate low I content of pasture and hay, but values considered healthy in some areas may be deemed goitre-producing in others	—	—	Iodine deficiency state due to lack of iodine in pasture or deficiency conditioned by unknown factors
Aphosphorosis	Cattle, and to lesser extent Sheep	Ditto	Principally S. Africa	Low blood inorganic P	Controlled by feeding P	Low P content	P fertilising beneficial	Can be produced experimentally on diets low in P but otherwise adequate	P deficiency state caused by lack of P in the pasture
Alkali Disease and Blind Staggers	Cattle, Horses, Sheep and Pigs	U.S.A. and Canada		Se found in tissues which do not normally contain Se and in blood of affected animals only	—	High content of Se in vegetation. Toxic forage plants 4 to 25 p.p.m. of Se. Toxic weeds several thousand p.p.m. of Se	—	Similar symptoms and lesions have been produced in rats, pigs and horses fed inorganic Se	Chronic and acute forms of Se poisoning due to ingestion of fodder containing abnormal concentrations of Se and seleniferous weeds
"Forage Anaemia" associated with Infectious Anaemia	Horses	Scandinavia and Finland	Forest pastures of N. Sweden, Norway and N. and E. Finland	—	Administration of yeast prevents appearance of symptoms of "forage anaemia"	High Mn content in hay and pasture. Limited data indicate low vitamin B_1 content of hay	Liming which reduces the availability of soil Mn to the plant has a beneficial effect	Symptoms of "forage anaemia" can be produced experimentally on vitamin B deficient diet and experimental animals show lowered resistance to anaemia virus. Administration of Mn, twice the amount in affected hay, produced symptoms of "forage anemia" in 1 of 2 horses	It is concluded that "forage anaemia" is a B_1 avitaminosis probably caused by oxidation of B_1 in pasture and hay in presence of large quantities of Mn. An additional effect may be caused by the effect of ingested Mn on vitamin B_1 metabolism. Such a vitamin B_1 deficiency predisposes to infectious anaemia
Oat Hay Poisoning	Cattle principally	U.S.A. and Canada		Methaemoglobinaemia	—	High KNO_3 content of hay, 3 to 7 per cent.	—	The condition has been produced in cattle, sheep and horses by feeding KNO_3	The disease is caused by ingestion of large amounts of nitrate which is changed into nitrite in the rumen and causes nitrite poisoning

* — signifies no information.

† Licking diseases have also been investigated in Schleswig-Holstein, other parts of Germany, Switzerland, the Dalarna district of Sweden and the coastal areas of Norway. The etiology has not been established, but there does not appear to be any sure foundation for the views that deficiency of Na or K or excess of K in relation to Na or low alkali-alkalescence have any causal connection with the disease. There is some evidence that in parts of Schleswig-Holstein deficiency of Cu may be a causal or at least a complicating factor

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